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Archives of Neurology and Psychiatry

VOLUME 25 JANUARY, 1931 NUMBER 1

ANASTOMOSIS OF NERVES

EXPERIMENTS IN WHICH THE CENTRAL END OF THE DIVIDED CERVICAL SYMPATHETIC NERVE WAS ANASTOMOSED TO THE PERIPHERAL END OF THE DIVIDED FACIAL NERVE AND TO THE PERIPHERAL END OF THE DIVIDED HYPOGLOSSAL NERVE *

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The earliest experiments on the anastomosis of nerves (1827) were those of Flourens.[1] He wrote:

> I divided in a cock the two main nerves which go from the brachial plexus, the one to the superior and the other to the inferior aspect of the wing. On section of the first of these nerves, the wing began to droop and to move with difficulty, on section of the second it drooped altogether; and its tip, the part chiefly supplied by the divided nerves, could no longer be moved at all. I then crossed the two divided nerves and united the central end of one with the peripheral end of the other, and maintained this artificial crossing with a stitch. After some months the bird had completely regained the use of the tip of its wing, which no longer drooped, and which he used in flying quite as well as before the experiment.
>
> A long time after complete cicatrization of the external wound I exposed the divided nerves and found that they had completely united, and that in the position in which I had placed them, that is to say, that the peripheral end of the one had united with the central end of the other. . . . When I pinched the central end of the superior nerve above the point of union the muscles on the inferior surface of the wing contracted, and on the other hand, when the inferior nerve was pinched above the point of union, the muscles on the upper aspect of the wing contracted. The communication of impulses was then perfectly reestablished throughout the whole length of the reunited nerves, and moreover its direction was crossed, the crossed direction being determined by the artificial crossing of the reunited nerves.

It appears obvious that as the result of the experiment some part of the central nervous system had to relearn its function, and that nerve fibers previously conveying impulses to extensor muscles subsequently carried impulses to a flexor group of muscles, and vice versa.

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* Submitted for publication, June 25, 1930.

* The experiments were performed at the National Institute for Research, Hampstead. Mr. Colledge assisted with some of the experiments. The late Sir David Ferrier was present at almost all of the experiments. The microscopic sections were cut, stained and photographed by Mr. Steward at the Royal College of Surgeons by permission of Sir Arthur Keith.

1. Flourens: Recherches expérimentales sur les propriétés et les fonctions du système nerveux, dans les animaux vertébrés, ed. 2, Paris, J. B. Baillière, 1842, chap. 17, p. 272.

Langley,[2] in 1898, published some experiments in which he had united the vagus and lingual nerves to the cervical sympathetic nerve. The central end of the cranial nerve in each case was united to the peripheral end of the cervical sympathetic nerve. Functional union took place.

> Stimulation of the *vagus* had a greater effect on the nictitating membrane, and on the arteries of the ear, than upon the pupil; and a greater effect upon the pupil, than upon the erector muscles of the face area. Stimulation of the *lingual* caused the ordinary effects produced by stimulating the cervical sympathetic except the dilatation of the pupil.

These effects were: (1) retraction of the nictitating membrane, (2) slight bulging of the eyeball, (3) separation of the eyelids, (4) slow contraction of the arteries of the ear, (5) a movement of a few hairs on the area of the face, but (6) no certain effect was obtained on the pupil.

Langley wrote: "There is no fundamental difference between the nerve fibers. The function of the peripheral nerve depends on the nerve ending." Flourens,[3] as long ago as 1827, contributed an essay on the "Unity of the Nervous System."

It should be remembered that the cervical sympathetic nerve is composed of white fibers, the medullary sheaths and axis cylinders of which stain in the same manner as do the cranial and peripheral nerves.

Langley and Anderson,[4] in 1904, anastomosed the central end of the fifth cervical nerve to the peripheral end of the cervical sympathetic nerve (superior cervical ganglion). The effects of stimulation of the fifth cervical nerve showed clearly that functional union had taken place between the two nerves, for all the usual effects of stimulation of the cervical sympathetic nerve were observed: (1) contraction of the arteries of the ear; (2) retraction of the nictitating membrane; (3) slight opening of the eyelids; (4) bulging of the eyeball; (5) slight erection of hairs on the facial area, and (6) dilatation of the pupil. Langley and Anderson reported that microscopic sections showed that some of the fibers in the superior cervical ganglion were larger than sympathetic fibers, but these did not extend beyond the upper part of the superior cervical ganglion.

An experiment was performed on a cat which confirmed and extended the results obtained by Langley. The central end of the divided cervicofacial division of the facial nerve was united to the peripheral end of the divided cervical sympathetic nerve. At the end

2. Langley: J. Physiol. **29**:241, 1898.

3. Flourens (footnote 1, chap. 12).

4. Langley and Anderson: J. Physiol. **30**:439, 1904.

of six months: 1. There was no enophthalmos. 2. There was no prominence of the nictitating membrane. 3. The pupil was not small, but was not quite so large as the opposite or normal pupil. 4. The whisker hairs on the side of the operation no longer drooped but stood out like those of the opposite side. 5. The muscles of the lower lip on the side of the operation remained paralyzed; these were atrophied, allowing dribbling of saliva on this side of the mouth.[5]

THE CONDUCTIVITY AND EXCITABILITY OF A NERVE

Some previous experiments [6] throw light on the problem. Conductivity and excitability are clearly different phenomena. When an

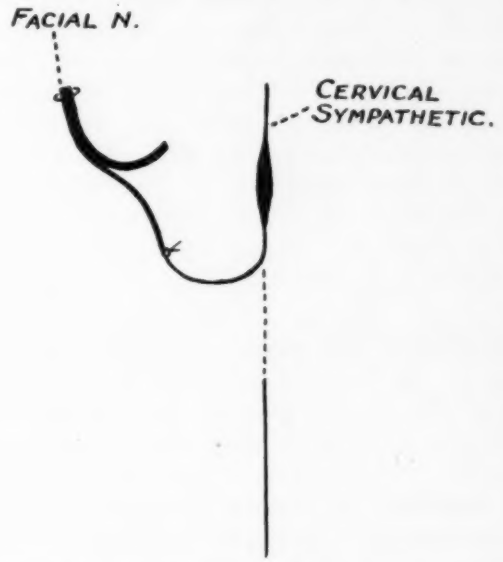

Fig. 1.—Diagram of the operation of facial (proximal segment) cervical sympathetic (distal segment) anastomosis.

opportunity offered for stimulating directly the nerves of the limb in double lateral implantation after functional recovery and before the death of the animal, a notable result was obtained. On faradic stimulation of the peripheral segments of the two nerves, it was found that a much stronger current was required to produce the same effect than was necessary when faradic stimulation was applied to the central segments of the two nerves; in other words, the same strength of current caused

5. The recovery recorded was seen by the late Sir David Ferrier, by Sir Charles Sherrington and by Dr. Dale.

6. Ballance, Charles; Colledge, L., and Bailey, L.: Further Results of Nerve Anastomosis, Brit. J. Surg. **13**:533, 1926.

less and less response as the stimulation proceeded from the central to the peripheral parts of the nerves.

This phenomenon has been described as the phenomenon of the avalanche.[7] There is in the experiment cited a clear differentiation between the phenomenon of conductivity and that of excitability. The difference in functional activity of the nerve fibers in the proximal and peripheral segments of the nerves appears to depend on a difference in histologic and anatomic structure; in other words, on the greater or less perfection of the process of regeneration attained by them. Durante referred to regenerating nerve fibers in which the function of conductivity is regained before that of excitability.

It is possible that an electric stimulus may be employed which is not suitable for the purpose of exciting in sympathetic fibers such an impulse as would cause contraction of striated muscle, or that the nervous impulse which is excited and which may cause contraction of unstriated muscle is the factor which makes a nerve fiber refractory to the excitation of an impulse which would cause contraction of striated muscle. "The refractory state is not merely a local effect at the point of application of the stimulus, but is the same at any point in the nerve after the passage of the propagated disturbance."[8] "Inhibition can be changed to excitation by strychnine."[9] It is unknown whether the nerve impulses required for excitation of voluntary and involuntary muscle are exactly identical. It is also unknown whether the central nervous system uses some process[10] different from that which is the basis of conduction by excitation in peripheral nerves, or whether a nervous impulse originating in the brain is in any way different from one arising in the peripheral nerve fibers as the result of artificial stimulation. "No structure is found at the periphery (of a sympathetic nerve) like end plates, but the nerve ends in arborisations round ganglion cells. Relays of fibers arise from these ganglion cells and pass to the unstriated

7. Durante: Phénomène de l'avalanche (Pfluger) de la boule de neige (Chauveau), in Cornil and Ranvier: Manuel d'histologie pathologique, ed. 3, Paris, Félix Alcan, 1907, vol. 3, p. 495. Durante pointed out that the functional restoration may occur ("transmission active de neuroblaste à neuroblaste: conductibilité plasmique") while there is still incomplete regeneration of the nerve fiber, and that it is important to distinguish the function of conductivity from that of excitability. He described the latter as follows: "pouvoir de transformer des vibrations (excitations) diverses en des vibrations nerveuses" (to be able to transform diverse vibrations [excitations] into nerve vibrations).

8. Bayliss: Principles of General Physiology, ed. 4, New York, Longmans, Green & Company, 1924, p. 390.

9. Bayliss (footnote 8, p. 435).

10. Lucas, Keith, and Adrian: The Conduction of a Nervous Impulse, New York, Longmans, Green & Company, 1915, p. 2.

muscle fibres."[11] "Under normal circumstances the excitatory impulse never arises in the fibres. The excitability of nerve fibres is rather a condition of their conductivity rather than an autonomous property."[12] "The greater the frequency of stimuli applied to the nerve may favour the absence of response in the muscle. Adrian was able to foretell the relation between the strength and frequency of stimulation which would give absence of contraction."[13] "Conduction with a decrement is a normal happening in certain parts of the nervous system. Conduction at the synapse may present the same features."[14] "Anaesthetics—ether and chloroform—temporarily abolish the current of action and the excitability of the nerve."[15] In the experiments related in this paper the animals were deeply anesthetized. "Fröhlich found that on anesthetising a tag of nerve the excitability diminished gradually while conductivity is at first unaltered, and when the excitability had fallen to a certain point, conductivity suddenly disappears."[16] "Alternating currents of high frequency and sufficient intensity to light an electric lamp have no stimulating action on nerve or muscle, probably because they paralyse conductivity."[17] These paragraphs, culled from recent physiologic writings, throw some light on the difficulties encountered in this research. The striking thing to note about the experiments described in this paper is that never before has the cervical sympathetic nerve[18] been called on to convey a nerve impulse suitable to energize the motor end-organs of striated muscle and so cause contraction of this type of muscular fiber.

The experiments described in this paper are the reverse of those described by Langley, in that the central end of the divided cervical sympathetic nerve was united to the peripheral end of the divided facial nerve and in another experiment to the peripheral end of the divided hypoglossal nerve. The object of the experiment in which the cervical sympathetic nerve was anastomosed with the hypoglossal nerve was to elucidate the apparently contradictory results obtained following ten experiments in which the central end of the cervical sympathetic nerve was united to the peripheral end of the trunk of the facial nerve.

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11. Starling: Principles of Human Physiology, ed. 4, Philadelphia, Lea & Febiger, 1926, p. 224.

12. Luciani: Human Physiology, English Translation, New York, The Macmillan Company, 1917, vol. 3, p. 259.

13. Lucas and Adrian (footnote 10, p. 91).

14. Lucas and Adrian (footnote 10, pp. 28 and 29).

15. Luciani (footnote 12, p. 212).

16. Luciani (footnote 12, p, 229).

17. Luciani (footnote 12, p. 223).

18. Cannon, W. B.; Binger, C. A. L., and Fitz, R. (Experimental Hyperthyroidism, Am. J. Physiol. **36**:363, 1914-15), united the phrenic nerve with the cervical sympathetic nerve in cats. The symptoms produced were comparable to those of exophthalmic goiter in man.

CERVICAL SYMPATHETIC-FACIAL ANASTOMOSES

These experiments were performed on six cats, two monkeys and two dogs. Six or more months after an experiment was done, and when the muscles of the face had recovered voluntary movement and gave a response to faradic stimulation, the cervical sympathetic was exposed in the lower part of the neck and stimulated with the faradic current. No contraction of the muscles of the face occurred. Stimulation of the cervical sympathetic-facial anastomosis and of the cervical sympathetic proximal to the anastomoses for a distance of 1¼ inches (3.1 cm.) did cause contraction of the muscles of

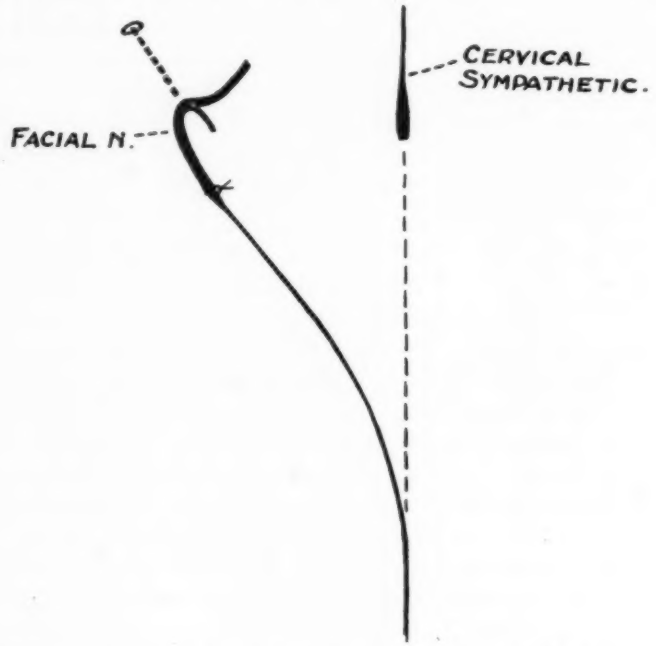

Fig. 2.—Diagram of the operation of cervical sympathetic-facial anastomosis. The sympathetic nerve is separated from the vagus till a point is reached just above the ganglion of the trunk of the vagus nerve. In the diagram the knife has been passed through the lower end of the superior cervical ganglion. The facial nerve trunk is divided at the stylomastoid foramen and gently turned forward. The fine needle and thread are then passed through the severed ends of the two nerves. They are thus brought together and the sutures tied. In only two experiments have ganglion cells of the superior cervical ganglion been demonstrated in the microscopic sections. Possibly in other experiments the cellular invasion at the site of union of the two nerves destroyed them. The experiments leave the impression that earlier recovery of faradic contractibility of the facial muscles occurs when the sympathetic nerve is divided than when the section takes place through the lower end of the ganglion. This may be so as some of the sympathetic fibers end their journey in these ganglion cells.

the face. In the operation of cervical sympathetic-facial anastomosis it is not possible to avoid a good deal of disturbance of the tissues of the neck, and thus under the sternocleidomastoid a considerable amount of scar tissue forms. Through the scar tissue the displaced fine sympathetic nerve becomes embedded. In two instances at autopsy long and careful dissection failed to follow and define this tiny nerve through the scar tissue.

Dr. Dale suggested that, as no movement of the facial muscles occurred when the cervical sympathetic was stimulated in the lower part

Fig. 3.—Drawing of site of anastomosis in cervical sympathetic-facial anastomosis; × 300. Cat; after twelve months. The nerve fibers twist and turn in every direction. The larger fibers are the facial fibers. They appear in short lengths as the section is in one plane while the fibers in their course occupy many planes. The smaller fibers are sympathetic fibers.

of the neck, facial nerve fibers had crossed in the scar tissue between the stylomastoid foramen and the site of the anastomosis. It was further suggested that they had grown down the sympathetic nerve for a distance of 1 inch (2.5 cm.), then turned upward and finally entered the two branches of the facial nerve. But no large facial fibers were seen in the microscopic sections of the sympathetic nerve proximal to the anastomosis, and other facts came to light which seemed to negative this explanation.

The experiment of uniting the facial nerve in a baboon to the cervical sympathetic nerve, to the glossopharyngeal nerve or to some other nerve of the neck is not strictly comparable to the operation of anastomosing the facial nerve in man to another nerve for the purpose of curing facial palsy. The division of the facial nerve in man at the stylomastoid foramen means division of a nerve no longer in active life and function. Destruction of the facial nerve by injury or disease in the canal of Fallopius had taken place a long time before, proximal to the site of the operative section. It was otherwise in the experimental

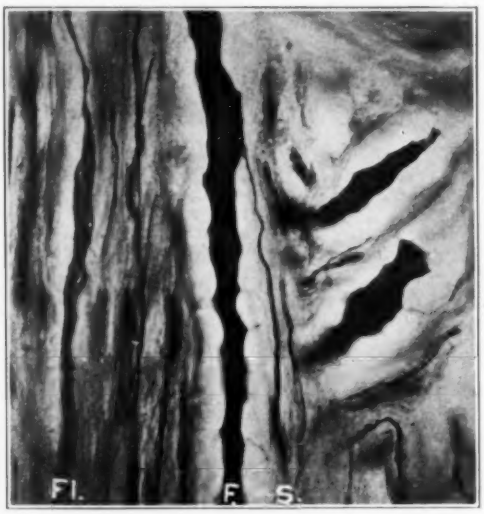

Fig. 4.—Photomicrograph of part of the same section as figure 3; × 1,000. *F* is a facial nerve fiber. The projections from its side can be seen to be connected by delicate bands with the inner aspect of the medullary sheath. The projections from the side of the facial nerve fiber are probably due to the attachment of the axis cylinder at regular intervals to the inner aspect of the sheath by means of very fine connective tissue trabeculae, while the intervening portions of the axis cylinders are depressed owing to shrinkage during the preparation of the specimen. Thus the trabeculae maintain the axis cylinders in the center of the sheath in a manner comparable to similar trabeculae in man which cross the subarachnoid space and sustain the brain stem in position. In the turtle similar trabeculae cross the subdural space, as in this animal the subarachnoid space is only a potential space. The aorta of the turtle is surrounded by a capacious lymph sheath and connective tissue bands cross the lymph space between the outer wall of the aorta and the inner surface of the lymphatic sheath. *S* is a sympathetic nerve fiber which joins the facial fiber. At the point of junction the sympathetic nerve fiber is expanded, and the expansion is not unlike the disk-like bodies seen on the terminal filaments of the axis cylinders of a motor end-organ. *Fl* is another facial fiber. There seems to be an end-to-end junction between the facial fiber and a small fiber. At this junction the microscope reveals clearly an enlargement.

operations in which at the moment of section the facial nerve was an actively living and functioning nerve. Notwithstanding the known formation of a bulb at the proximal end of a severed nerve, it appeared possible that in some instances nerve fibers had reunited the divided ends of the facial nerve.

Fig. 5.—Drawing of part of the same section as in figures 3 and 4; × 1,200. The larger fiber in a sheath is a facial fiber. The fine fibers within the sheath are presumably sympathetic fibers. Some of the fine fibers are seen to join the large fiber.

The results of the experiment were as follows:

1. In one experiment on a cat, in which recovery of the muscles of the face had taken place, the stylomastoid foramen was exposed, and

all the scar tissue filaments in this region were divided. No effect was observed subsequently in the condition of the muscles of the face.

2. In one experiment on a cat in which the muscles of the face had recovered:

(*a*) Stimulation of the sympathetic in the lower part of the neck produced no movement in the muscles of the face.

(*b*) Stimulation of the sympathetic for 1 inch proximal to the anastomosis caused contraction of the muscles of the face (Dr. Dale).

(*c*) The brain was removed and the facial nerve stimulated at the internal auditory meatus. Movements of the pinna but no contraction of the muscles of the face occurred. The posterior auricular nerve had

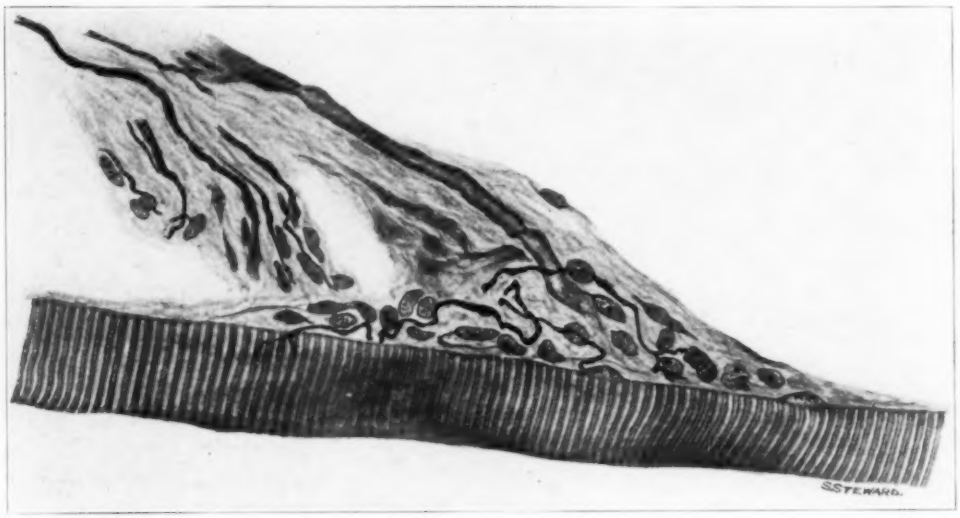

Fig. 6.—Drawing of a motor end-organ in the orbicularis oris muscle of a dog, after eight months. Cajal stain; × 500. The end-organ is seen edge-wise. The nerve as it approaches the end-organ undergoes division, probably repeated division, and at last the terminal fibers enter a bed of granular matter beneath the sarcolemma. This granular matter contains a number of cells in which the very fine axis cylinders terminate. The chromatin of the nuclei of these cells presents an irregular figure, and it is in this darkly staining material that the terminal filaments of the axis cylinders end. Other terminal filaments of the axis cylinders (see fig. 8) end in ramifications which appear on the side of a muscle fiber as oval or round plates. It is easier to show the course of the axis cylinders in a drawing than in a photograph, as the photograph shows only one level, while a drawing may be a composite representation of several levels. The end-organ and the muscle fiber are normal.

not been divided at the operation. The posterior auricular nerve leaves the facial nerve just within the canal as a rule and not outside the stylomastoid foramen.

(*d*) No nerve fibers were seen in the microscopic sections of the scar tissue between the bulb on the facial nerve and the oval swelling at the site of the cervical sympathetic facial anastomosis.

3. In a baboon in which the muscles of the face had recovered and in which at the operation the trunk of the facial nerve was noted as only one-eighth inch (0.3 cm.) in length:

(*a*) Stimulation of the sympathetic in the lower part of the neck produced no contraction of the muscles of the face.

(*b*) Stimulation of the sympathetic at the anastomosis and for a short distance proximal to the anastomosis produced slight contractions of the muscles of the face.

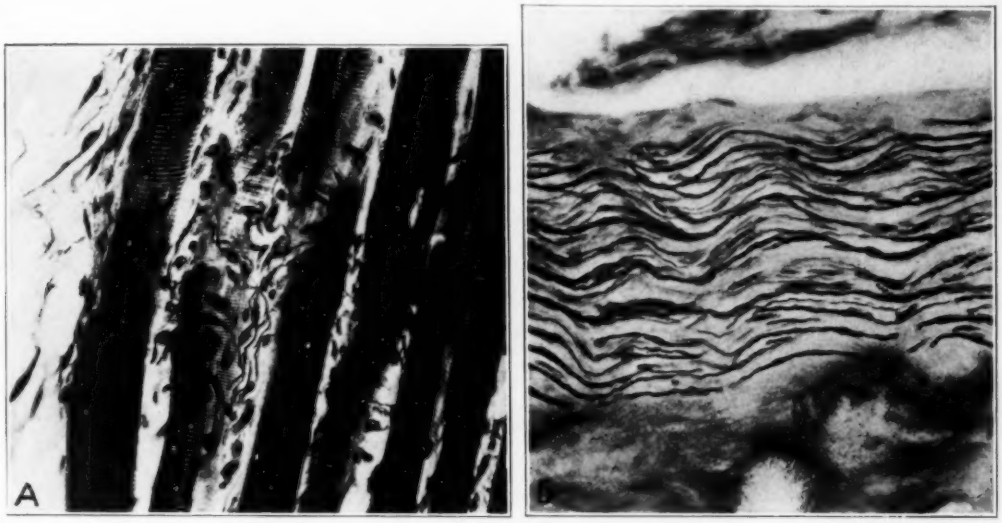

Fig. 7.—*A*, photomicrograph of motor end-organs in the orbicularis oris muscle of the dog; × 300. Note the disklike terminal expansions of the axis cylinders. *B*, photomicrograph of a regenerated nerve just before it enters the orbicularis oris muscle of the dog; × 300.

(*c*) The brain was removed, and the facial nerve, stimulated at the internal auditory meatus, caused contractions of the muscles of the face but the contractions were not nearly so powerful as the contractions caused by stimulation of the facial nerve at the internal auditory meatus on the opposite or normal side.

(*d*) The microscopic sections showed nerve fibers in the scar tissue between the bulb on the facial and the site of the cervical sympathetic-facial anastomosis.

It would appear therefore that:

1. In some of the experiments a partial reunion of the ends of the facial nerve took place. Unfortunately, the brain was not removed at

the conclusion of each of the experiments and the facial nerve stimulated at the internal auditory meatus. It is therefore not possible to report in how many cases this reunion of the end of the facial nerve occurred. It is certain that they did not do so in all the experiments, and that the predisposing factor was the shortness of the trunk of the facial nerve.

2. In one experiment, at least, no reunion of the ends of the facial nerves occurred.

3. The scar tissue underneath the sternocleidomastoid muscle prevented the passage of a nerve impulse when the sympathetic in the lower part of the neck was stimulated with the faradic current. Clinical

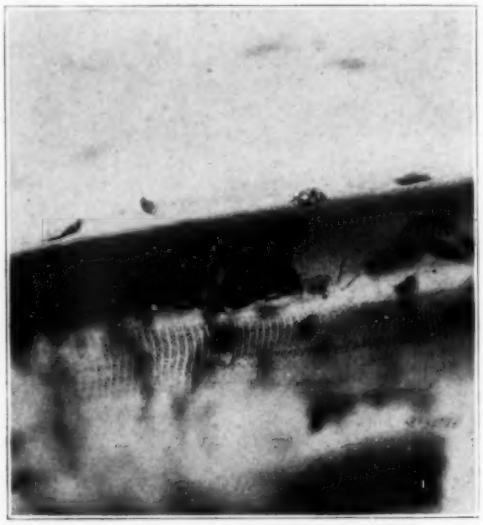

Fig. 8.—Photomicrograph of a motor end-organ in the occipitofrontalis muscle; × 650. Figure 7 and this figure show that the end-organs, muscles and nerves are healthy. It will be seen that some of the terminal filaments of the axis cylinders vary in thickness and end at the side of the muscle fiber in round or oval diskshaped expansions which have been described as giving the appearance of a bunch of berries.

experience seems to point to the fact that a nerve impulse originating in the central nervous system may pass while the attempt to induce artificially by faradism a nerve impulse to travel along a peripheral nerve embedded in scar tissue may fail.

The General Effects Observed in a Typical Experiment on the Cat, After Varying Periods of Time, Following Cervical Sympathetic-Facial Anastomosis.—These are complex since both the facial and the sympathetic nerves have been divided. In consequence of the doubt indicated

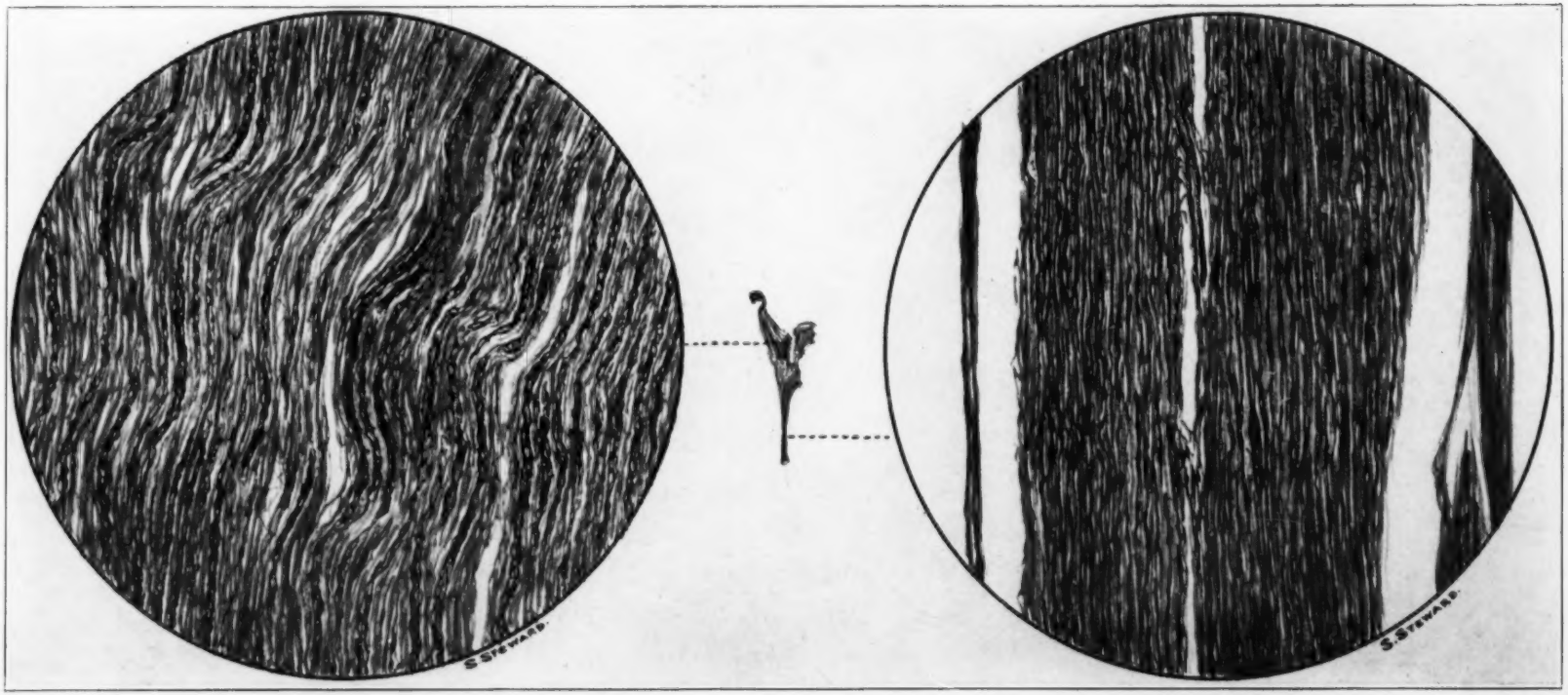

Fig. 9.—Drawings of Weigert-stained sections of the facial and sympathetic nerves (cat, after ten months), to show the different size of the fibers of the two nerves: facial fibers, 0.01 mm. thick; sympathetic fibers, 0.005 mm. thick. The circle on the left shows facial fibers; × 250; that on the right, sympathetic fibers; × 250.

previously as to the cause of the recovery of the facial muscles, these observations have less value than they would otherwise possess.

At First: There is enophthalmos, small pupil and other signs of division of the sympathetic. These signs persist. The face is flaccid and paralyzed, the eyelids do not close, and dribbling of saliva may occur for seven or ten days.

In Three Months: The face is symmetrical, and on stroking the bristles the upper lip is raised and the eyelids close; so recovery of the neuromuscular mechanism has already reached a stage in which a reflex act is possible.

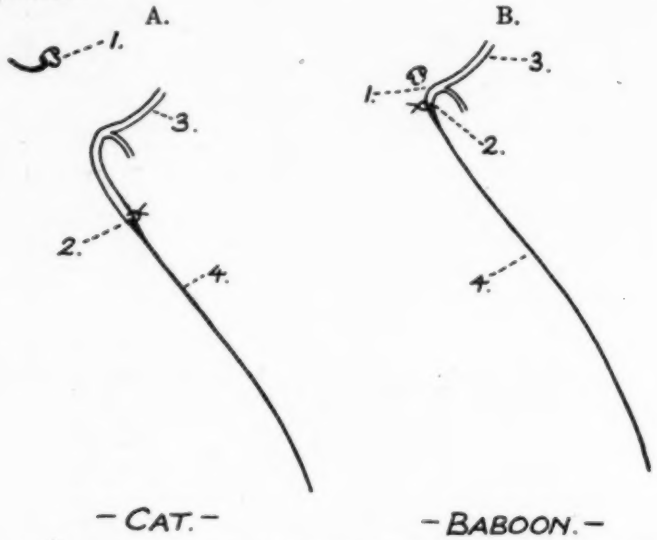

Fig. 10.—*A*, cat: usual distance of divided nerve at stylomastoid foramen from anastomosis: *1*. Bulb on proximal end of divided facial. The nerve passing backward from it is the posterior auricular nerve (fig. 11 *A*). *2*. Anastomosis (fig. 11 *B*). *3*. Temporofacial nerve (fig. 11 *C*). *4*. Sympathetic. *B*, baboon: trunk of facial nerve unusually short, causing the distance between the stylomastoid foramen and the anastomosis to be much diminished: *1*. Space between bulb of facial and site of anastomosis (fig. 13 *A*). *2*. Anastomosis (fig. 13 *B*). *3*. Temporofacial nerve (fig. 13 *C*). *4* Sympathetic.

In Four Months: There is no deformity of the face at rest. On eating, however, less movement is seen on the previously paralyzed side of the face than on the normal side. On faradic stimulation with a moderate current, all the muscles respond well; the least movement takes place in the occipitofrontalis muscle.

In Seven Months: The face is symmetrical during eating. On faradic stimulation, all the muscles respond well as compared with those of the normal side.

In Another Experiment: When the electrode is placed just behind and above the angle of the mouth, the eyeball advances and the eyelids separate. This must be due to contraction of the nonstriated orbital muscle of Müller. The superior cervical ganglion had not been removed

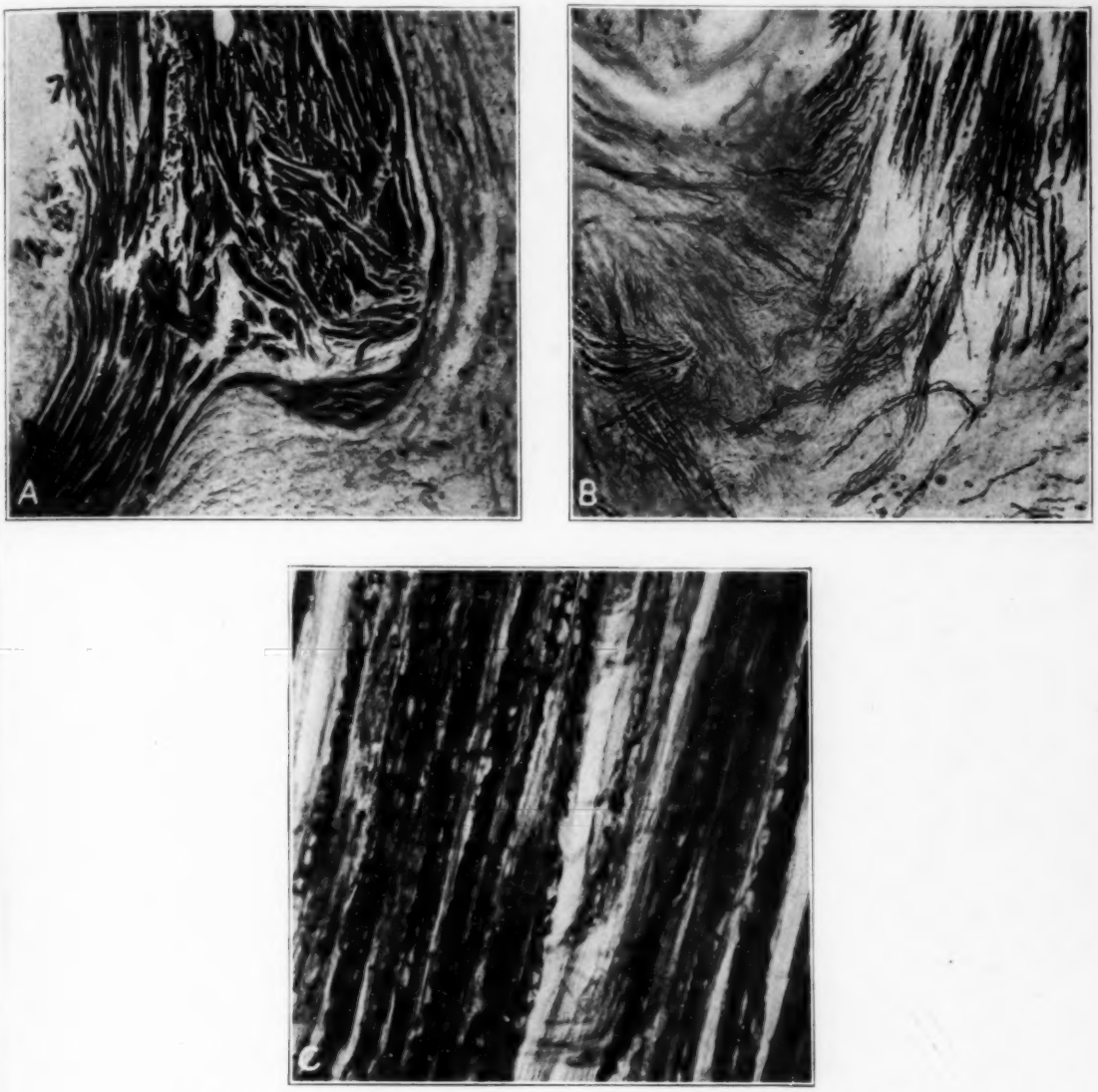

Fig. 11.—*A*, see figure 10 *A*, *1*. *B*, see figure 10 *A*, *2*. *C*, see figure 10 *A*, *3*.

and hence the muscle of Müller presumably has remained unatrophied. In some experiments the pupil dilated and then contracted, but as the dilatation was not an invariable happening it may not have been due to the faradic stimulation. The animals were under deep ether anesthe-

sia, and dilatation of the pupils occurs under such anesthesia. This movement of the eyeball does not occur till the muscles of the face respond to direct faradic stimulation through the skin of the face, and

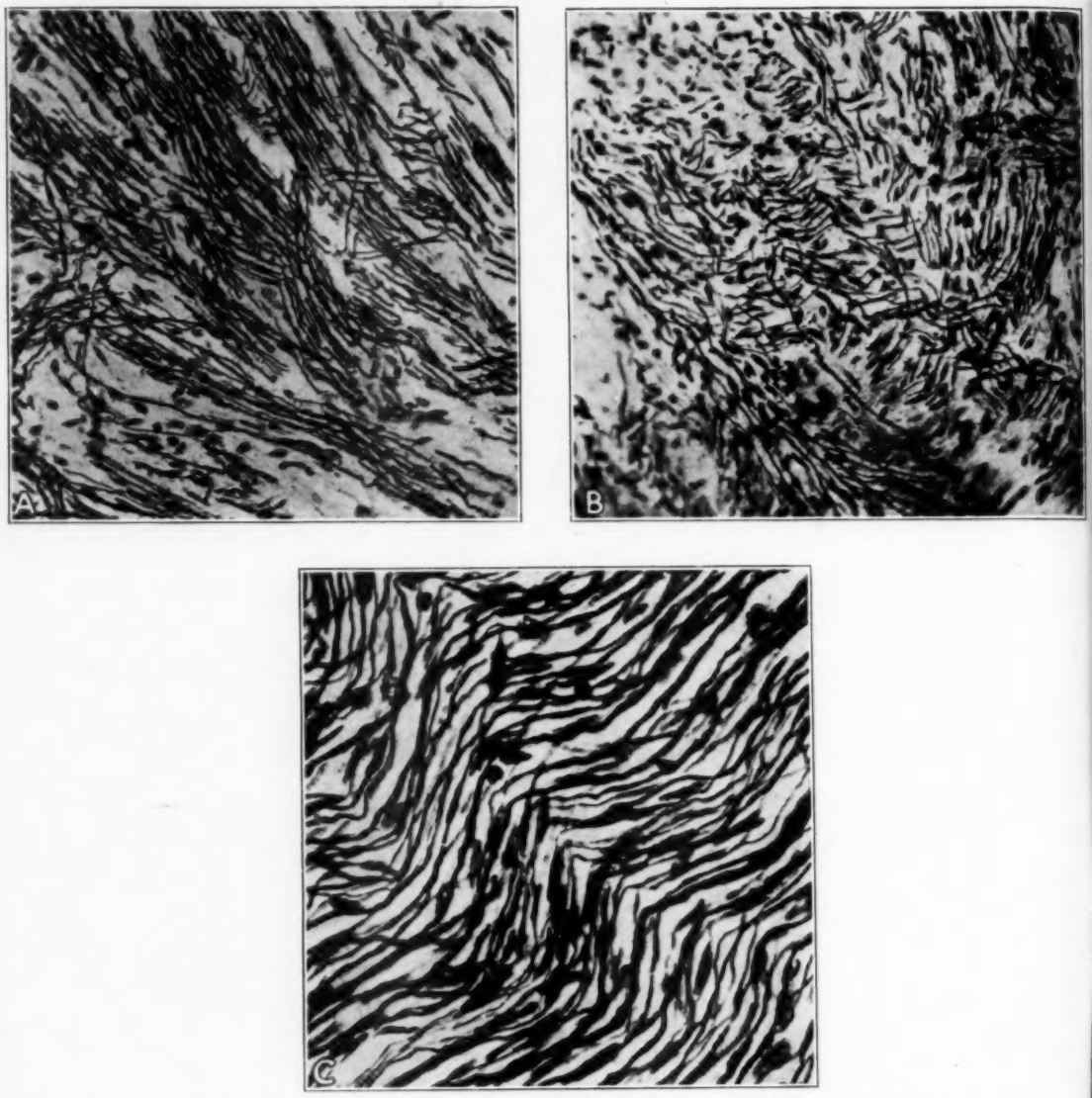

Fig. 12.—*A*, see figure 10, *B*, *1*. *B*, see figure 10 *B*, *2*. *C*, see figure 10 *B*, *3*.

it does not occur on the normal side of the face. It is not easy to offer an explanation of the phenomenon.

In Nine Months: In one experiment the appropriate area of the cerebral cortex was stimulated. Before doing this the stylomastoid

foramen was exposed, and all the scar tissue filaments in the region of the foramen were divided. All the muscles of the face contracted; Sherrington's electrode was used.

The effects of stimulation of the cervical sympathetic proximal to the anastomosis have already been described.

It was then determined to unite the cervical sympathetic nerve to another cranial nerve. The choice fell on the hypoglossal nerve. The following experiment was performed on a cat.

Operation 1: Cervical sympathetic-hypoglossal anastomosis, end-to-end.

Operation 2: Cervical sympathetic-descendens hypoglossi anastomosis, end-to-end.

The hypoglossal nerve was divided proximal to the origin of the thyrohyoid branch. It was freed as far as the base of the skull, divided a second time and removed. The sympathetic nerve was divided below the ganglion, and the proxi-

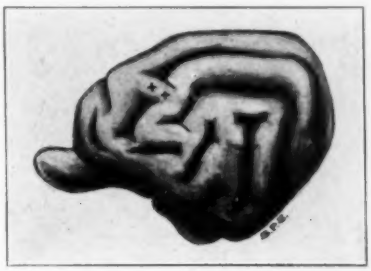

Fig. 13.—Lateral view of the left side of a cat's brain, after nine months. The area at which stimulation of the cortex cerebri caused movements of the muscles of the opposite or right side of the face is indicated by crosses. This area was in the extreme anterior part of the gyrus suprasylvius medius and just above the upper end of the sulcus coronalis. The sulcus coronalis separates the gyrus coronalis in front from the gyrus suprasylvius anterior behind. In the middle of the gyrus coronalis is the adduction center for the vocal cords (Semon and Horsley), and in the middle of the gyrus suprasylvius anterior is the abduction center for the vocal cords (Semon and Horsley: Phil. Tr., vol. 181B). The names of the sulci and gyri are taken from Winkler and Potter: Anatomical Guide to Experimental Researches on the Cat's Brain.

mal end united to the distal cut end of the hypoglossal nerve. The descendens hypoglossi nerve was divided at the same time that the long piece of the hypoglossal was excised. The distal cut end of the descendens hypoglossi was sutured to the anastomosis already carried out between the cervical sympathetic and the hypoglossal nerves.

The results of the operation were as follows:

Three Months After the Operation.—The animal was etherized and the tongue examined.

1. The right half of the tongue (the side of the operation) was thinner and less in width than the left half of the tongue; a groove was seen down its center.

2. The whole of the right side of the tongue gave a response to faradic stimulation, but with a current stronger than was required to cause a similar contraction on the left side.

Six Months After the Operation.—The tongue was examined.

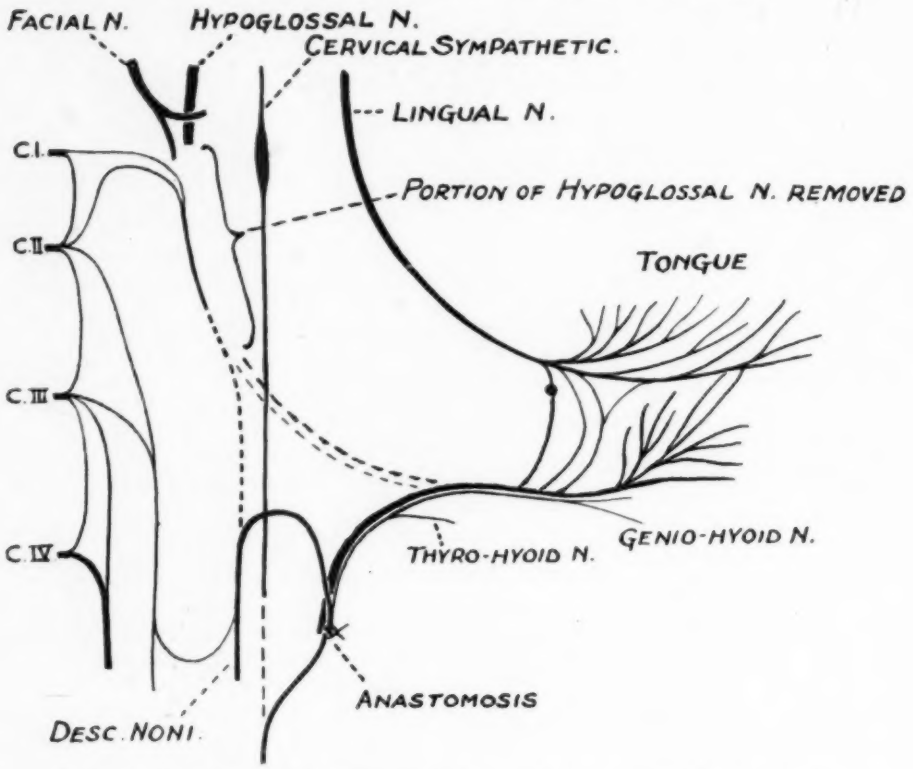

Fig. 14.—Diagram of cervical sympathetic-hypoglossal anastomosis and cervical sympathetic-descendens hypoglossi anastomosis.

1. The right half of the tongue had recovered its size and form as compared with the left side. When the mouth of the cat was opened, the tongue retracted; both sides of the tongue appeared to move together.

2. The right half of the tongue under faradic stimulation gave a response equal to that of the left side with the same strength of current.

3. On stimulation of the cervical sympathetic in the lower part of the neck, a ripple of muscular contraction occurred along the right half of the tongue (Dr. Dale).

4. The sternohyoid and sternothyroid muscles did not appear on inspection in any way different from those of the opposite side. When these muscles were stimulated directly with the faradic current, a normal response was obtained. When the cervical sympathetic was stimulated in the lower part of the neck, their behavior was not noted.

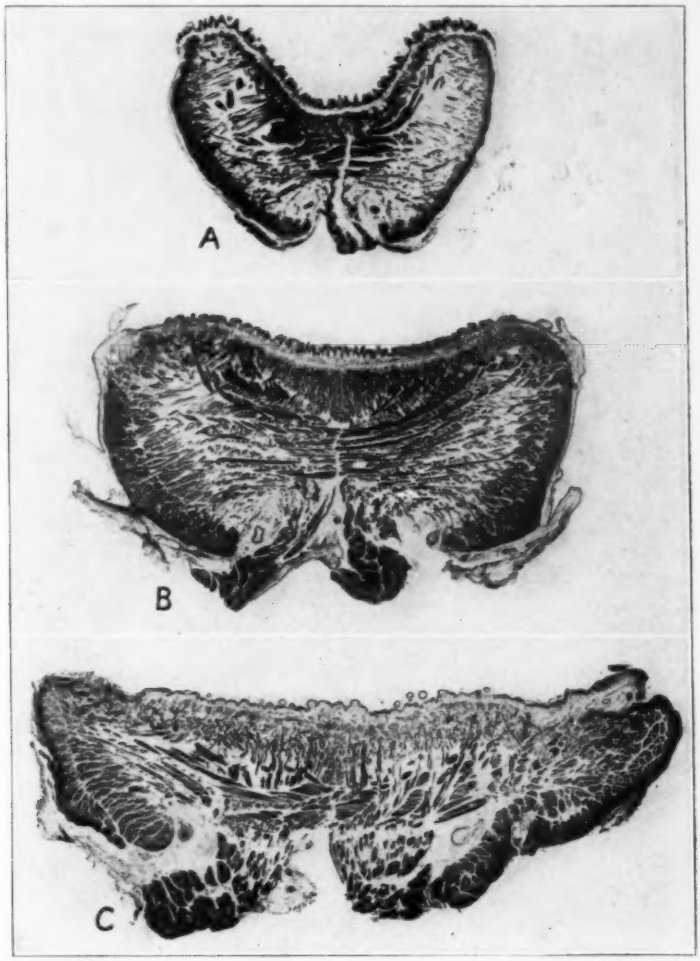

Fig. 15.—Cervical sympathetic-hypoglossal anastomosis; after six months. Recovery of tongue; transverse section; × 3. *A*, front of tongue; *B*, middle of tongue; *C*, back of tongue. The section of the back of the tongue is somewhat irregular; this is probably due to the pinning out on cork during hardening.

5. The usual signs associated with division of the cervical sympathetic nerve persisted.

6. The operation of cervical sympathetic-hypoglossal anastomosis causes very little disturbance of the tissues of the neck as compared

Fig. 16.—Recovery of transverse muscle of tongue; × 450.

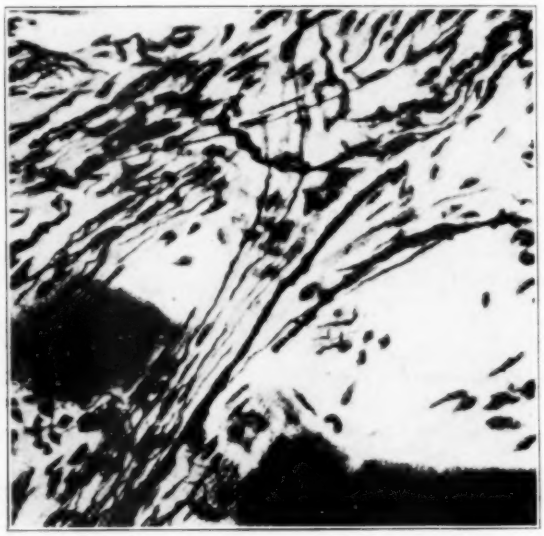

Fig. 17.—Site of anastomosis; × 450.

with the operation of cervical sympathetic-facial anastomosis. Hence the resulting scar tissue is much less.

7. The removal of the hypoglossal nerve from the base of the skull to the site where the thyrohyoid branch is given off made it impossible for the two ends of the hypoglossal to reunite. Further dissection showed that no such reunion had taken place.

8. As I was in North America for three months before the final examination of this cat was undertaken, the action of the tongue of the cat in the lapping of milk was not observed. This is unfortunate.

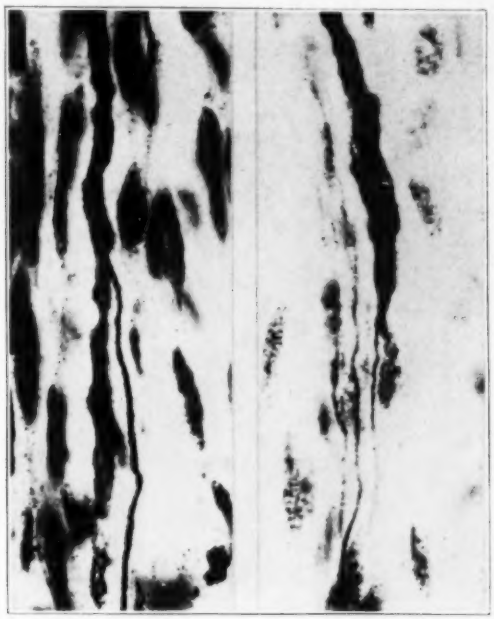

Fig. 18.—Apparent junctions of sympathetic and hypoglossal fibers at site of anastomosis; × 1,000.

The operation of cervical sympathetic-hypoglossal anastomosis was followed by recovery of the muscles of the tongue. The operation of cervical sympathetic-descendens hypoglossi anastomosis was followed by recovery of the sternohyoid and sternothyroid muscles. These experiments appear to answer the question as to whether the cervical sympathetic nerve is competent to carry nerve impulses to the nerve endings in striated muscular fiber. Just as recovery of the intrinsic muscles of the larynx follows recurrent laryngeal-phrenic anastomosis, so does recovery of the muscles of the tongue follow cervical sympathetic-hypoglossal anastomosis, and so does recovery of the sternohyoid and sternothyroid muscles follow cervical sympathetic-descendens hypo-

glossi anastomosis. The essential element in the neuromuscular mechanism is not a particular group of nerve fibers. The essential elements are the central nerve cells and the nerve endings in the muscular fibers.

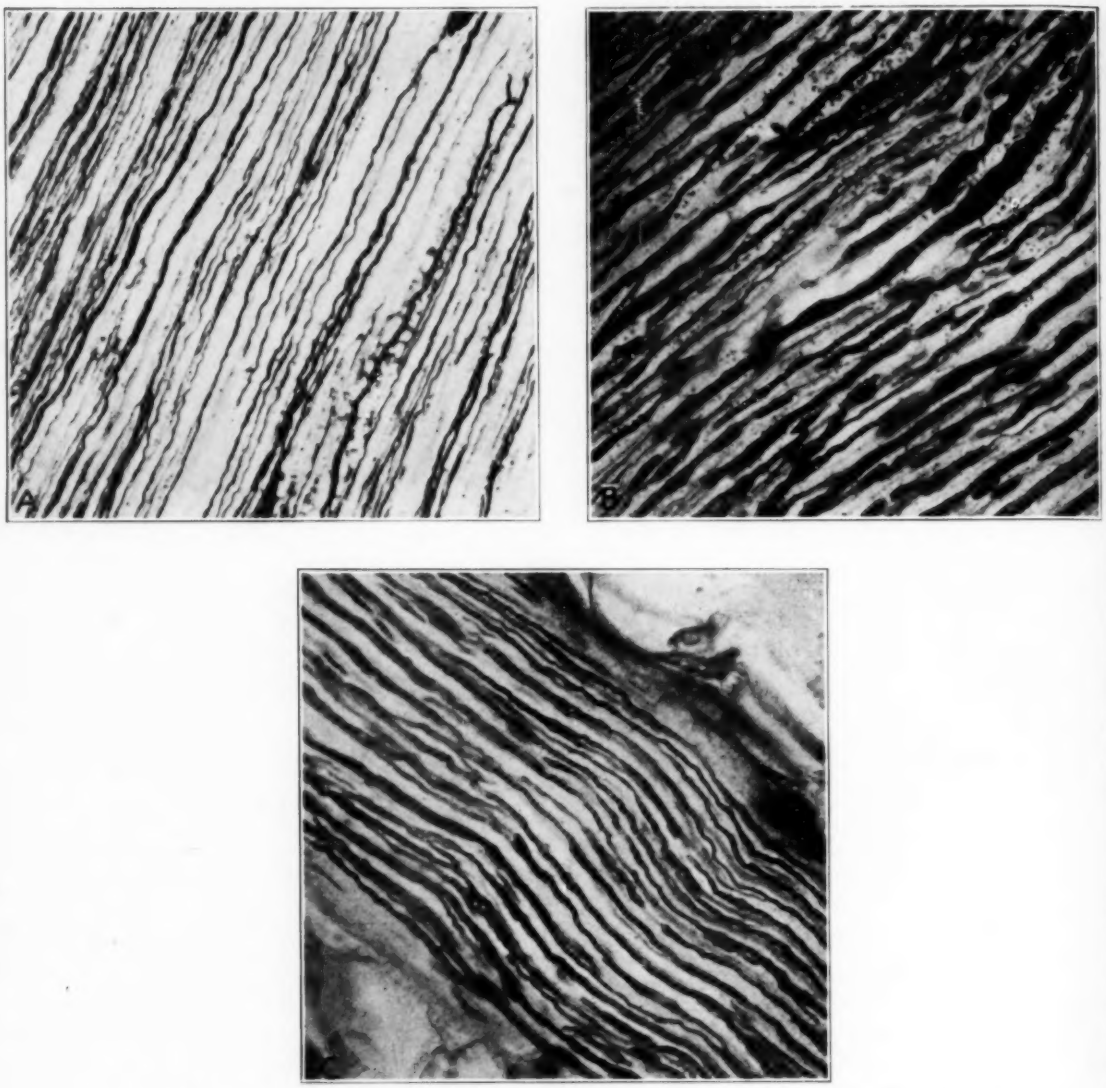

Fig. 19.—*A*, right hypoglossal nerve near but distal to the site of anastomosis; × 450. *B*, right hypoglossal just before entering tongue; × 450. *C*, left hypoglossal (on the normal side) just before entering the tongue; × 450.

It is possible to travel from New York to San Francisco by several routes though one route is the more common and the more direct route

than the others. Under the experimental conditions related, the nerve impulses do not travel by the ordinary routes.

In the case of a recurrent laryngeal-phrenic anastomosis the nerve impulses travel via the phrenic route because the route via the vagus is closed. In the case of a cervical sympathetic-hypoglossal anastomosis, the nerve impulses travel via the cervical sympathetic instead of the normal route via the hypoglossal nerve; and in the case of the cervical sympathetic-descendens hypoglossi anastomosis, the nerve impulses reach the motor end-organs of the striated fibers of the sternohyoid and sternothyroid muscles via the cervical sympathetic nerve instead of through the descendens hypoglossi nerve.

Fig. 20.—Recovery of right sternohyoid muscle; × 450.

Lastly, the cervical sympathetic nerve having been employed for anastomosis with the facial, hypoglossal and descendens hypoglossi nerves, the question arises, Do these experiments throw any light on the sympathetic innervation of striated muscle? Hunter's remarkable experiments on birds are highly suggestive of a dual innervation of muscle. Prof. Elliot Smith, in his critical address to the Section of Neurology, Royal Society of Medicine, on Feb. 11, 1926, summed up in the following words: "One thing which comes out clearly from all this experimental work is that section of the sympathetic nerves renders the appropriate group of muscles much more liable to fatigue than the normal muscle. The muscle which is fatigued as the result of the sympathetic denervation becomes definitely toneless."

Kulchitsky [19] figured a dual innervation of the muscle fibers of the python by nonmedullated and medullated nerve fibers. He stated that the nonmedullated and medullated fibers never terminate in the same muscle fibers; that the motor end-plate of the nonmedullated nerve fiber has not the granular substance which is so characteristic of the motor end-plate of the medullated fiber, and that it lies outside the sarcolemma while the motor end-plate of the medullated nerve fiber lies within the sarcolemma.

As regards the experiments related in this paper, complete functional and anatomic recovery of the striated muscles of the face, of the tongue and of the depressor muscles of the hyoid bone and larynx were observed. No muscle fibers of the face or tongue muscles or of the sternohyoid or sternothyroid muscles appeared to be atrophied as they should be if a dual innervation was necessary for complete restoration of function. The sympathetic nerve fibers seem to join at the anastomoses with the larger cranial nerve fibers and there is no evidence in the microscopic sections that they continued into the cranial nerve trunks.

It is difficult to come to any conclusion as to whether these experiments give support to the view that the anatomic and functional perfection of striated muscle depends on a dual innervation, but I am inclined to think that they do.

While I was staying in Cairo, Dr. Wilson, Emeritus Professor of Physiology in the University of Cairo, contributed the following remarks:

I take it that the aim of the experiments is to show the following:

1. That regeneration of the motor nerve to a striated voluntary muscle may be brought about by union with the central cut end of an autonomic nerve such as the cervical sympathetic.

2. That after regeneration electrical excitation of the sympathetic will give rise to normal contraction in the striated muscle.

3. That the natural stimuli arising in the central origin of the sympathetic will restore the nutrition and tone of the striated muscle.

4. That these natural stimuli originating in the sympathetic will give rise to contractions in the muscle supplied by the anastomosed nerve.

5. That when regeneration is complete coordinated movements of the muscles on the operated and unoperated sides take place normally, although in the former the central path has been entirely changed.

These seem to be the questions for which an answer may be sought in the results recorded. The answer to the first four seems to be definitely in the affirmative. The fifth, which a little consideration will show to involve a much more remarkable fact than the first four, may be thought to require some further experimental proof, although it seems to be demonstrated if the results are correctly interpreted.

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19. Kulchitsky: J. Anat. **59**:1 (Jan.) 1924.

In considering these experiments, the following general principles may be stated as representing the view which would be accepted in regard to the cerebrospinal somatic nerves on the one hand or the autonomic system on the other. Whether they apply equally to the conditions produced by suture of an autonomic to an efferent somatic motor nerve, has not hitherto been investigated and seems to be the fundamental question dealt with.

1. A nerve acts as a conductor; if efferent the result of excitation depends on the nature of the peripheral organ; if afferent, on the central termination.

2. The health of a muscle and its recovery from the state of atrophy into which it would fall after section of its nerve are dependent on its maintenance in a condition of some sort of activity by stimuli reaching it through the nerves from the central nervous system. This state of activity may be either: (*a*) tonic, continuous but variable in degree, or (*b*) active contractions. Whether these are really separate forms of activity depending on a double nerve supply (sympathetic-cerebrospinal) implying a difference in the character and origin of the impulses reaching the muscle is still uncertain. The experiments recorded seem to have a bearing on this question, at any rate so far as they show that impulses originating in the central origin of the sympathetic are appropriate to the maintenance of tone in a striated muscle.

3. Functional coordinated movements of groups of muscles, such as those of the face or tongue, are dependent on messages starting from the proper area of the cerebrum, conveyed by certain paths to the nuclei of origin of the motor nerves and distributed thence by the motor nerves to the respective muscles.

Langley's observations referred to in this paper prove that the anastomosis of the preganglionic part of either type of autonomic nerve was equally effective: that, in fact, the impulses traveling along either were interchangeable, at any rate so far as these were produced by electrical excitation of the nerves. It is to be remembered that the impulses carried by the anastomosed nerves in his experiments were conveyed not to the peripheral organs but to the ganglion cells from which arose the post-ganglion fibers actually supplying the tissues.

Langley's observations do not seem to show conclusively that the natural stimulus arising in a nucleus of one nerve is appropriate to the excitation of all the functions of the tissues supplied by another nerve. Nor do they show that an autonomic nerve can be effectively anastomosed to the motor nerve to a striated muscle. Langley, in fact, definitely assumed that the fibers of the vagus supplying striated muscle would not be appropriate to the formation of an effective anastomosis with the sympathetic.

It is evident that the question "Can a stimulus applied to any nerve united to a second nerve excite the normal response at the peripheral distribution of the second nerve"? is not the same as the question "Will the natural stimulus from the central origin of a nerve give rise to the normal response at the periphery when this nerve is cut and united to a second nerve having quite a different central origin?"

Although there are differences in the most effective stimuli for different types of nerve fibers, the first question might be accepted as probable for an anastomosis between any types of efferent nerves.

In regard to the second this seems to be true of two motor nerves to voluntary muscle and also, as demonstrated by Langley, between two autonomic nerves. It might, however, be doubted *a priori* whether if (*a*) were sympathetic and (*b*) somatic motor the natural stimulus arising in the origin of (*a*) would be appropriate to the excitation of the function of (*b*). Then experiments appear to show, however, that this is so.

I am not quite sure whether in these experiments, apart from the restoration of the tone of the muscles there was, under conditions when a sympathetic reaction, such as dilatation of the pupil, would have been expected, contraction of the muscles of the face and tongue respectively in the two sets of experiments occurred. The centers of origin of the sympathetic are constantly emitting impulses which maintain the tone, vasomotor or other, of the tissues supplied. It is possible that such may be sufficient to maintain the nutrition and tone of the striated muscles after the anastomosis.

The conclusions one is bound to come to so far from the results recorded appear to be:

1. That an effective anastomosis can be made between the central end of the sympathetic and a motor nerve to voluntary muscle. The regenerated fibers seem to be of the large type characteristic of the motor nerve. This seems to agree with your views regarding the process of regeneration.

2. Stimulation of the sympathetic gives rise to normal contractions of the striated muscle.

3. The natural stimulus originating in the sympathetic origin is effective in maintaining the nutrition and tonic function of striated muscle. This applied also to the striations and other characters of the fibers. Unless therefore a double nerve supply (Hunter) exists to all the muscle fibers this can only mean that the regenerating fibers originating in the sympathetic were distributed to the normal nerve endings in the muscle fibers. This would make it unnecessary to expect to get the drug reactions, characteristic of the sympathetic terminations, from the muscles supplied by the anastomosed nerves.

The fifth question referred to at the beginning of this commentary, namely, whether coordinated movements of the muscles supplied by facial and hypoglossal respectively are reestablished after suture with the sympathetic is certainly a very remarkable fact if it can be regarded as conclusively demonstrated.

There seems to be no doubt that when a motor nerve, such as the median, after section is reunited and regeneration has taken place the correct movements of the various muscles are reestablished.

There is a "selective redistribution" of the impulses carried by the regenerated fibers and the muscles become correctly linked up to the center.

Langley demonstrated this definitely in his anastomosis of the divided cervical sympathetic. He showed by excitation of the spinal roots that the redistribution of the impulses to their proper destination was peripheral and not due to any rearrangement in the center. The selection, however, in this case was not in the distribution of the nerve fibers to the tissue but at the termination of the preganglionic fibers around the cells of the superior cervical ganglion, synapses being correctly formed around the appropriate cells. Langley suggests as the only feasible explanation, a chemiotactic influence of the nerve cells on the regenerating fibers.

It seems evident that this implies a corresponding difference in the nerve fibers or of the impulses (? rhythm) passing along them.

In such cases, whatever view may be taken as to the mechanism of selective distribution, the question is relatively simple as the central mechanism, both cerebral and spinal, remains intact.

I do not know whether it is conclusively proved that in a crossed anastomosis of two nerves going respectively to the flexor and extensor muscles of a limb the normal response can ultimately be reestablished. This seems to be analogous to the anastomosis between the hypoglossal (central) and the facial (peripheral)

with ultimate restoration of normal responses in the facial muscles. In either case the central origins are near together and form a part of the same functional system.

It is possible to understand the process of relearning by the laying down of relatively simple new paths.

Turning to the actual experiments recorded: In the sympathetic-facial anastomosis there appears to have been complete correspondence after regeneration was established of the movements on the operated and normal sides.

The objection to accepting the result as proving that the motor impulses governing these movements originated in the sympathetic is that in some of the experiments there seems to be evidence of the possibility of facial fibers having entered from the central stump of the nerve. This certainly seems unlikely in others of the experiments. I am not quite sure to what extent it was possible in any of the experiments to initiate contractions of the facial muscles by electrical stimulation low down in the neck. In some this was not possible although it was shown to occur on stimulation of the sympathetic above the scar tissue in the neck some way central to the point of anastomosis.

That the scar tissue pressing on the nerve could so alter the conductivity of the fibers that natural stimuli were transmitted but that the type of impulse aroused by direct excitation was blocked is possible, I cannot offer an opinion on this point. You would know from your experience of nerve suture whether it might be regarded as probable.

On the other hand, if the scar tissue completely blocked the passage of any kind of impulse this would be shown by the complete degeneration of the sympathetic fibers in the part of the sympathetic peripheral to the scar formation. If the supply was really by facial fibers these must have grown down the sympathetic for a considerable distance (in which case they would appear as large calibre fibers) and then turned back toward the periphery. Whether this is possible depends on the nature of the process of nerve regeneration. It seems improbable.

In regard to the sympathetic-hypoglossal experiment there seems to be no question of contamination with fibers originating in the stump of the hypoglossal. No objection is possible on this ground.

In this experiment the structure, health and tone of the muscles supplied by the hypoglossal were completely restored, and it may be assumed that as feeding became normal the action of these muscles on the operated and unoperated sides was normal and synchronized. Strictly speaking this is an assumption as although the two sides of the tongue when at rest appeared normal no observation is recorded as to exactly what happened during activity.

Assuming, however, that full functional control of the muscles supplied by the facial and hypoglossal respectively occurred, this assumption implies that impulses from the cerebral centers which normally should have reached their destination via the medullary nuclei of those nerves, found a new path by which they ultimately reached the cells of origin of the cervical sympathetic in the thoracic cord. It is difficult to understand the formation of such a new path or how, if it were formed, the selective distribution to particular muscles takes place.

It might be suggested that the corresponding movements of the two sides of the tongue were due to the fact that there may be normally sympathetic discharges which synchronize with the muscular contractions; for example, vasomotor impulses. It, however, is very difficult to suppose that such impulses could if they reached the striated muscle fibers give rise to the rapid and complex movements of the tongue.

If Hunter's view of the double innervation of striated muscle is correct the muscles must have their representation in the central origin of the sympathetic.

A muscle when acting must receive impulses through both its motor nerve and through the sympathetic supply. If this is so there must normally exist a path through which impulses can reach the muscles supplied for example by the hypoglossal. This might provide the way round which is required to explain the result of anastomosis of the sympathetic and hypoglossal after section of the latter nerve.

I have analyzed the various points raised as carefully as possible in order to determine what if any are the difficulties in accepting your interpretation of the results of these experiments. The last point referred to seems the only one that does present any serious difficulty. It is, however, of such interest and importance that it should be confirmed by further experiments even if the slight element of doubt mentioned be disregarded.

I am greatly indebted to Dr. Wilson for the interest he has taken in my work and for his promise to repeat the experiments. While maintenance of the tone and nutrition of a muscle is admitted to be the function of the sympathetic, it is not possible to imagine a muscle without activity to be a normal state. Tone and nutrition cannot alone produce a normal striated fiber. Activity is a part of the being of a striated fiber. It follows then that the complete health of a striated muscle fiber requires that impulses must come from the cerebral cortex as well as from the cells of origin of the sympathetic.

It is interesting to conjecture what would have happened in the experiment of sympathetic-hypoglossal anastomosis if the ciliospinal center in the medulla or cord had been stimulated. If the muscles of the tongue had contracted it would seem that Dr. Wilson's suggestion as to the pathway of the cerebral nerve impulses under the altered circumstances caused by the anastomosis would be correct. It would be necessary also to stimulate the appropriate area of the cortex.[20]

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20. Other papers on nerve anastomosis are: Ballance, Charles: Results Obtained in Some Experiments in which the Facial and Recurrent Laryngeal Nerves Were Anastomosed with Other Nerves, Brit. M. J. **2**:349 (Aug.) 1924. Colledge, L., and Ballance, C.: Anastomosis Between Recurrent Laryngeal and Phrenic Nerves: Conditions of Intrinsic Muscles and Nerves of Larynx of Rhesus Monkey More Than Three Years and Two Years After Operation, Brit. M. J. **1**:746 (May 5) 1928. Ballance, Charles: Some Results of Nerve Anastomosis, Brit. J. Surg. **11**:327 (Oct.) 1923; Experiments on Conduct and Fate of Ligature Made from Parietal Peritoneum of Ox When Implanted in Living Tissue, Lancet **2**:10 (July) 1926. Ballance, Charles, and Barnes, E. B.: Anastomosis of Recurrent Laryngeal to Phrenic Nerves: Some Recovery of Function, Brit. M. J. **2**:158 (July) 1927. Ballance, Charles; Colledge, L., and Bailey, L.: Further Results of Nerve Anastomosis, Brit. J. Surg. **13**:533 (Jan.) 1926.

PATHOLOGIC CHANGES IN PARALYSIS CAUSED BY DRINKING JAMAICA GINGER *

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In the spring of 1930, there occurred in Ohio, Kentucky, Alabama,[1] Mississippi,[2] Massachusetts [3] and other states an epidemic of peripheral neuritis. The patients gave histories of drinking commercial extract of ginger. The cases were characterized by an insidious development of foot drop and, somewhat later, of wrist drop. It is estimated that at the height of the epidemic there were about 500 cases in the Cincinnati district alone, while the combined admissions to the Cincinnati General Hospital and to St. Mary's Hospital numbered 264. A somewhat similar epidemic occurred in 1900 in Manchester, England, in which the toxic agent was determined to be arsenic arising from dextrose made from starch by the use of crude sulphuric acid containing the poisonous metal. The dextrose was used in the production of beer, the consumption of which brought about the widespread attack of peripheral neuritis.[4]

It was learned that for the past ten years extract of Jamaica ginger has been used by laborers as a substitute for whisky. Jamaica ginger extract is sold in two ounce bottles in small confectionery and grocery stores. It is usually taken by mixing it with a bottle of one of the carbonated waters. As soon as the cases of paralysis began to appear poisonous stocks were withdrawn, and investigators found it difficult to gather sufficient numbers of samples to make satisfactory analyses. It is altogether probable, therefore, that the toxic agent producing the paralysis may remain a matter of doubt.

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* Submitted for publication, July 16, 1930.

* From the Department of Anatomy (Neurology), University of Cincinnati Medical School.

1. Harris, S.: Jamaica Ginger Paralysis, South. M. J. **23**:375 (May) 1930.

2. Bennett, S. R.: Patients Suffering from Paralysis Due to Drinking Jamaica Ginger, South. M. J. **23**:371 (May) 1930.

3. Merritt, H. H., and Moore, M.: Peripheral Neuritis Associated with Ginger Extract Ingestion, New England J. Med. **203**:4 (July 3) 1930.

4. Reynolds, E. S.; Mann, J. D., and others: A Discussion on Peripheral Neuritis in Beer Drinkers: Its Precise Causation and Diagnosis, Brit. M. J. **2**:1044 (Oct. 12) 1901. Row, N.; Barendt, F. W., and Warrington, W. B.: Epidemic Arsenical Poisoning Amongst Beer Drinkers, Brit. M. J. **1**:10 (Jan. 5) 1901.

Andress [5] made analytic tests for cresol, phenol, various alkaloids and diethylphthalate. His studies tend to eliminate cresol, phenol and the alkaloids as possible causative agents. Studies with diethylphthalate are still in progress. Foulger [6] found probable evidence of arsenic as the toxic factor. Kehoe,[7] by analyses of urine and feces in a group of cases, eliminated the possibility of lead as an exciting agent.

While the toxic agent was not postively determined, it seems reasonable to assume that it most probably was a denaturant, and that the extract of Jamaica ginger was prepared by using some form of denatured alcohol instead of pure ethyl alcohol.

In most cases the patient complained of dull, aching pains in the calves of the legs, occurring from two to ten days after drinking extract of Jamaica ginger, but in some cases a longer time elapsed before symptoms were noted. The condition then gradually progressed. The patient noticed inability to walk properly, and complete foot drop and finally wrist drop developed. Occasionally there were nausea, vomiting and diarrhea. On physical examination, the wrist drop and foot drop were noted. There was at times a slight loss of light touch and pain sensibility, the involvement tending to correspond to the distribution of the radial and anterior tibial nerves. Examination of the cranial nerves in general revealed no abnormalities. The triceps, biceps and patellar reflexes showed no striking abnormalities. Blood counts and blood pressure were within normal limits. Examination of the spinal fluid showed no striking characteristics.

Nine cases in which a diagnosis of Jamaica ginger poisoning was made came to autopsy. Of this group, one case was uncomplicated by any other severe pathologic factor. This case is described in detail. In the other eight cases complicating factors entered which might possibly have obscured the essential pathologic changes. Three cases of the latter group will be noted briefly.

REPORT OF AN UNCOMPLICATED CASE

CASE 1.—*History.*—M. M., a woman, aged 59, was admitted to St. Mary's Hospital on March 27, 1930. She stated that in December, 1929, she was induced by a grocer to take Jamaica ginger extract for a disturbance in the stomach, and that since that time she had taken a bottle daily, except on Saturday and Sunday, when she usually took from two to three bottles. On March 15, 1930, she began to notice weakness in the legs, with some cramping pain in the calves. This gradually became more severe, and on March 27, complaining of inability to walk or to use her hands, she was admitted to St. Mary's Hospital.

5. Andress, F. J.: Personal communication to the author. The writer is connected with the Department of Preventive Medicine, University of Cincinnati.

6. Foulger, J. H.: Personal communication to the author. The writer is connected with the Department of Pharmacology, University of Cincinnati.

7. Kehoe, R. H.: Personal communication to the author. The writer is connected with the Department of Physiology, University of Cincinnati.

Physical Examination.—The patient was emaciated, but was mentally alert. The heart revealed no abnormalities. The blood pressure was 142 systolic and 82 diastolic, and the pulse rate, 92. The lungs and abdomen were normal. The Wassermann reaction of the blood was negative with all antigens. The urine was normal. The cerebrospinal fluid showed a faint trace of globulin, 2 cells, a negative gold curve and a negative Wassermann reaction. Stereoscopic roentgen studies of the lungs revealed no abnormalities.

A neurologic examination revealed: The pupils of the eyes reacted to light and distance; there was no disturbance of smell or ocular movement; the fundi were clear; common sensation over the face was normal; the masseters acted normally; there was no facial weakness; sensation of taste was present over the entire extent of the tongue; there was no disturbance of hearing, and the palatal responses were sluggish. The patient was able to speak, but she tired quickly. She was able to swallow solid foods with difficulty. The rate of respiration was 20. The tongue protruded somewhat slowly and in the midline. The patient was unable to use the feet or the hands; there was a bilateral foot drop and wrist drop, with more pronounced weakness in the feet. The biceps, triceps, wrist, patellar, achilles and abdominal reflexes were present. There were no pathologic reflexes. Because of the weakness in the hands and feet, tests for coordination could not be satisfactorily performed. There was only a slight diminution of pain and light touch sensation in the hands and feet, the borders of which were indistinct. The hands and feet were bathed in thin perspiration.

Course.—The patient's condition gradually grew worse. She developed incontinence of the bladder, followed in a few days by incontinence of the rectum. She gradually lost the ability to talk and to swallow solid foods, and later had extreme difficulty even in swallowing liquids. Respiration became difficult. The patient died on May 3, of respiratory paralysis.

Postmortem Examination.—An autopsy, performed by Dr. K. V. Kitzmiller, three hours after death, revealed emaciation with no abnormalities of the skin. The superficial group of lymph nodes were not palpable. The peritoneal cavity had a smooth, glistening lining and contained no free fluid. The liver was small; the cut surface had a slightly glazed appearance and distinct lobular markings. The gallbladder contained approximately 20 cc. of bile; its lining was unchanged. The spleen was small with a slightly thickened capsule and firm pulp; there was a slight increase in trabecular markings. The kidneys were small with some invasion of adipose tissue into the pelves; the capsules stripped with ease, leaving a slightly granular, deep red external surface; the cut surface presented some narrowing of the cortex; the sectioned vessels were somewhat thickened; the pyramids were well outlined. The renal pelves, ureters and urinary bladder presented a normal appearance. The uterus and adnexa were not remarkable. The suprarenal glands were of average size. The gastro-intestinal tract, including the pancreas, presented no evidence of abnormality. The lungs were free in the pleural cavity, except for a few adhesions at the apex of the upper lobe of the left lung; there was some scarring of the tissue, but elsewhere crepitus was normally present through all of the lobes. The bronchial tree, as well as the peribronchial lymph nodes, showed nothing abnormal. The pericardial cavity contained a few cubic centimeters of straw-colored fluid; the lining was smooth and glistening. The heart was small; the ventricles were contracted; the epicardium and the sectioned myocardial surfaces appeared normal. The valve leaflets and cusps were soft and freely movable. Early atheromatous changes were noted in the aorta, particularly in the arch. The coronary vessels possessed smooth glistening linings.

Gross Examination of the Brain.—On removing the calvarium, the dura was found to be slightly congested. On exposing the brain, a large collection of serous fluid was noted in the subarachnoid space. The brain was examined after fixation in formaldehyde. The blood vessels at the base showed no evidence of abnormality, and the membranes at the base were clear and not abnormal. There was a cone of swelling in the cerebellum in an area immediately surrounding the medulla. The convolutions were flattened and the fissures narrowed. There was a moderate degree of venous congestion. The pia-arachnoid membrane over the cortex was slightly clouded. A horizontal section through the brain revealed no abnormality aside from a number of minute cysts in the choroid plexuses of the lateral ventricles.

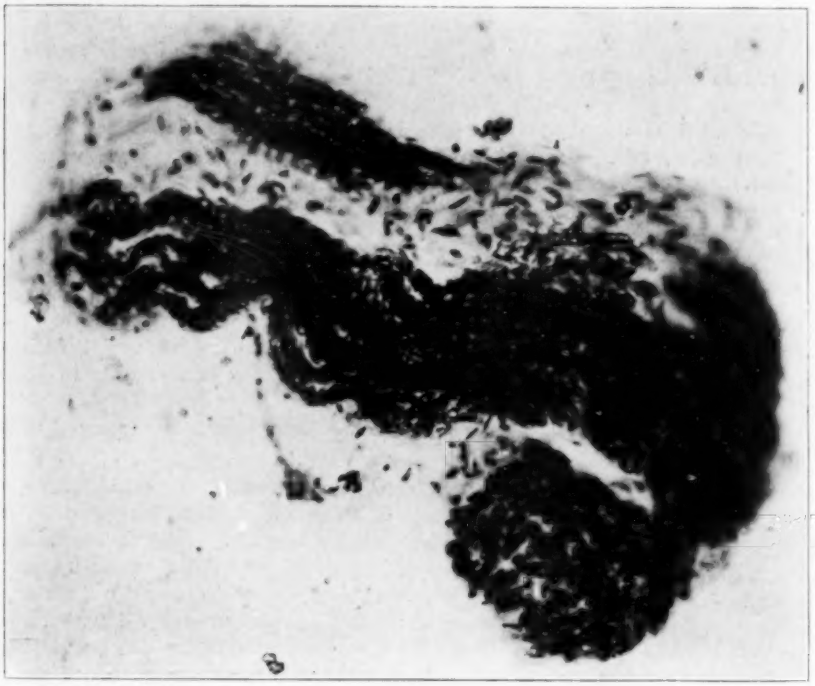

Fig. 1.—Section through the anterior root of the lumbar spinal cord. Note the degeneration of fibers near the center of the specimen.

Histologic Observations.—Portions of the radial and anterior tibial nerves, cervical and lumbar areas of the spinal cord, the entire medulla and the caudal portion of the pons were placed in 10 per cent commercial formaldehyde and embedded in celloidin. Serial sections were made of the entire medulla and caudal portions of the pons. The sections were stained according to Morgan's method for demonstrating changes in the cell body and degeneration of the fiber;[8] the celloidin sections were cut 34 microns thick, placed in 4 per cent ferric ammonium sulphate as a mordant, rinsed in water, stained in 1 per cent

8. Morgan, L. O.: Personal communication to the author. The writer is connected with the Department of Anatomy, University of Cincinnati.

aqueous hematoxylin, decolorized with 1 per cent aqueous hydrochloric acid, neutralized with a solution of sodium bicarbonate, cleared in carbol xylol from 95 per cent alcohol, followed by xylol, and mounted in balsam.

Microscopic examination of the radial nerve revealed a scattered loss of myelin, and degenerative changes with an absence of cellular infiltration (fig. 2). The anterior tibial nerve revealed similar scattered amyelinization, with an absence of an inflammatory type of cellular reaction. The study of cross-sections

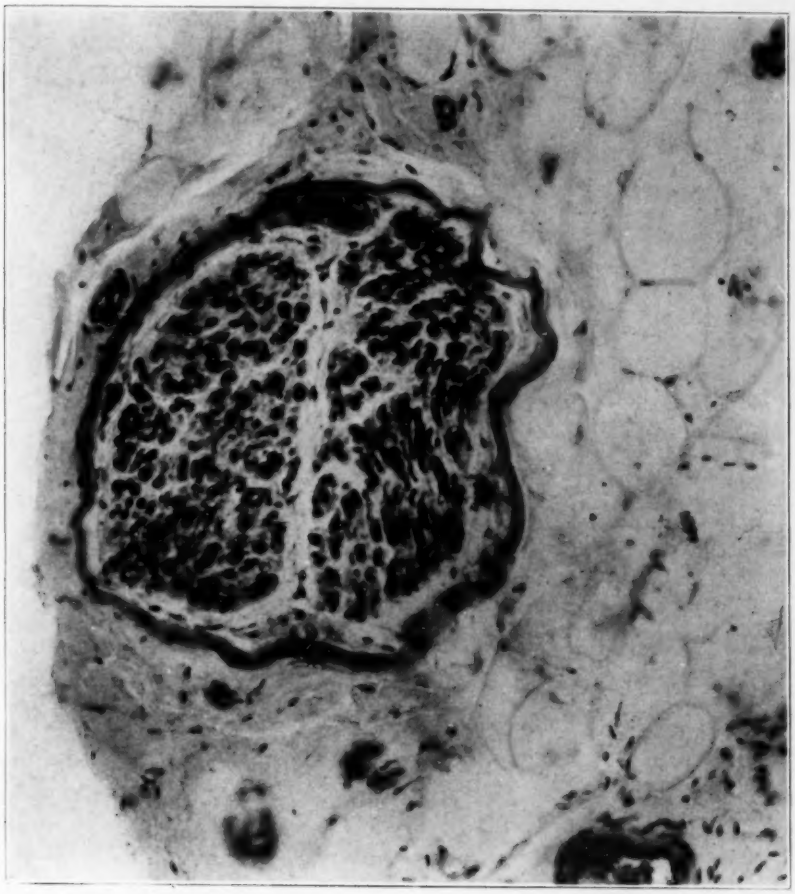

Fig. 2.—Cross-section of a portion of the radial nerve. Note degeneration of myelinated fibers.

of the lumbar spinal cord revealed normal blood vessels and pia-arachnoid membrane. The anterior root fibers showed marked degeneration in some bundles while other bundles remained intact (fig. 1). The fibers of the posterior roots showed no pathologic changes. The fiber tracts of the white matter showed no striking changes. The anterior horn cells of the lumbar spinal cord were, in large part, swollen, with mild and severe chromatolysis and eccentric nuclei (fig. 3). Central chromatolysis was the predominating type of pathologic change (fig. 4).

There were some shadow cells. There were occasional heavily stained and severely shrunken cells. There was no perivascular infiltration or neuronophagia. There did not appear to be any proliferation of glia cells. The spinal cord was diffusely infiltrated with amyloid bodies. Sections through the cervical enlargement of the spinal cord revealed changes which were strikingly similar to those of the lumbar cord. There were degenerative changes with loss of myelin in

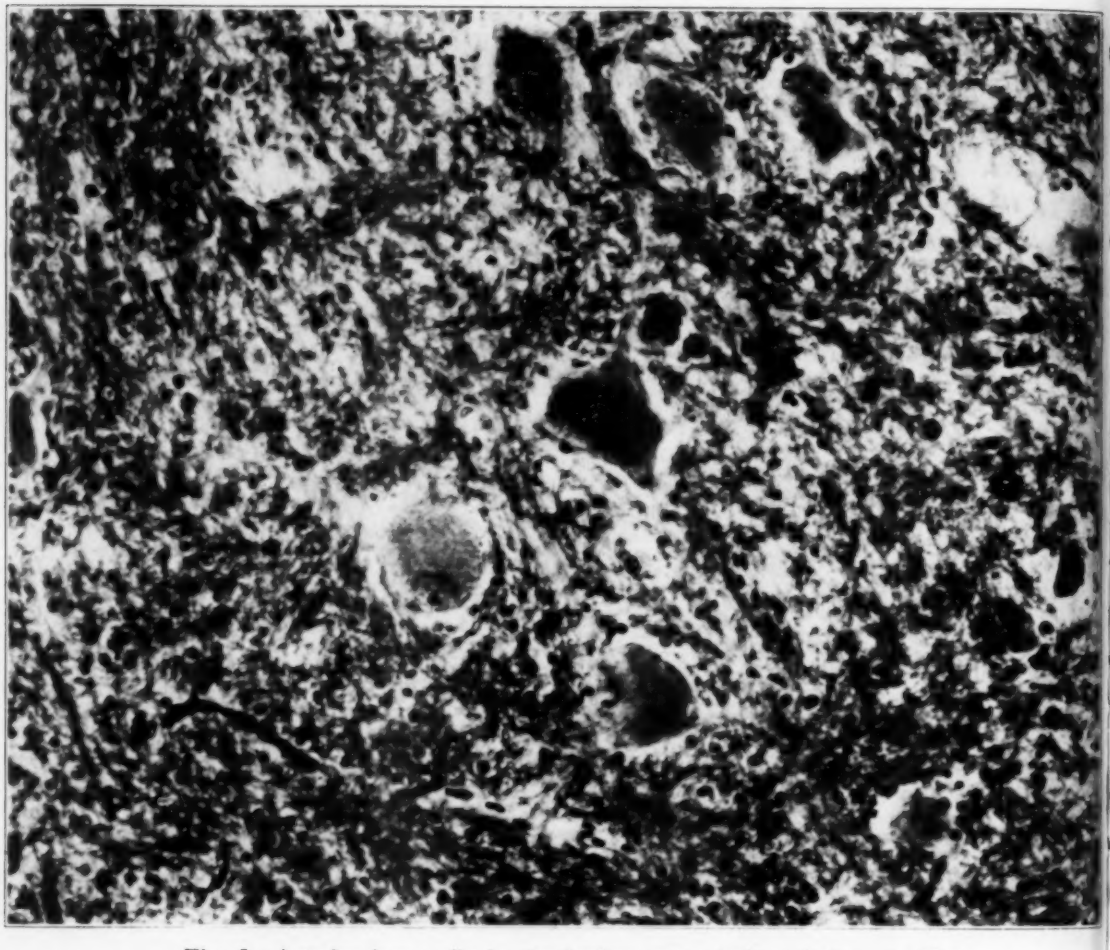

Fig. 3.—Anterior horn cells in the lumbar area of the spinal cord. Note chromatolysis, swelling, eccentric nucleus and the absence of reactive cells. Heavily stained circular areas are amyloid bodies.

the anterior root fibers, while the posterior root fibers remained intact. The pia-arachnoid membrane showed no evidence of abnormality. The fiber tracts of the white matter were intact. The anterior horn cells showed swelling, eccentric nuclei, chromatolysis and even death of the cells, without evidence of phagocytic cellular reaction, either endothelial leukocytes, phagocytic glia cells or polymor-

phonuclear leukocytes (fig. 5). This area of the cord was likewise diffusely infiltrated with amyloid bodies. Serial sections through the entire medulla and caudal portion of the pons revealed an intact pia-arachnoid membrane and normal blood vessels. Bundles of fibers in the root of the vagus nerve were degenerated (fig. 7). All of the areas of the medulla and the pons were heavily infiltrated with amyloid bodies. The myelinated fiber systems appeared to be intact. Chromatolysis with swelling, eccentric nuclei and occasional shadow cells were noted

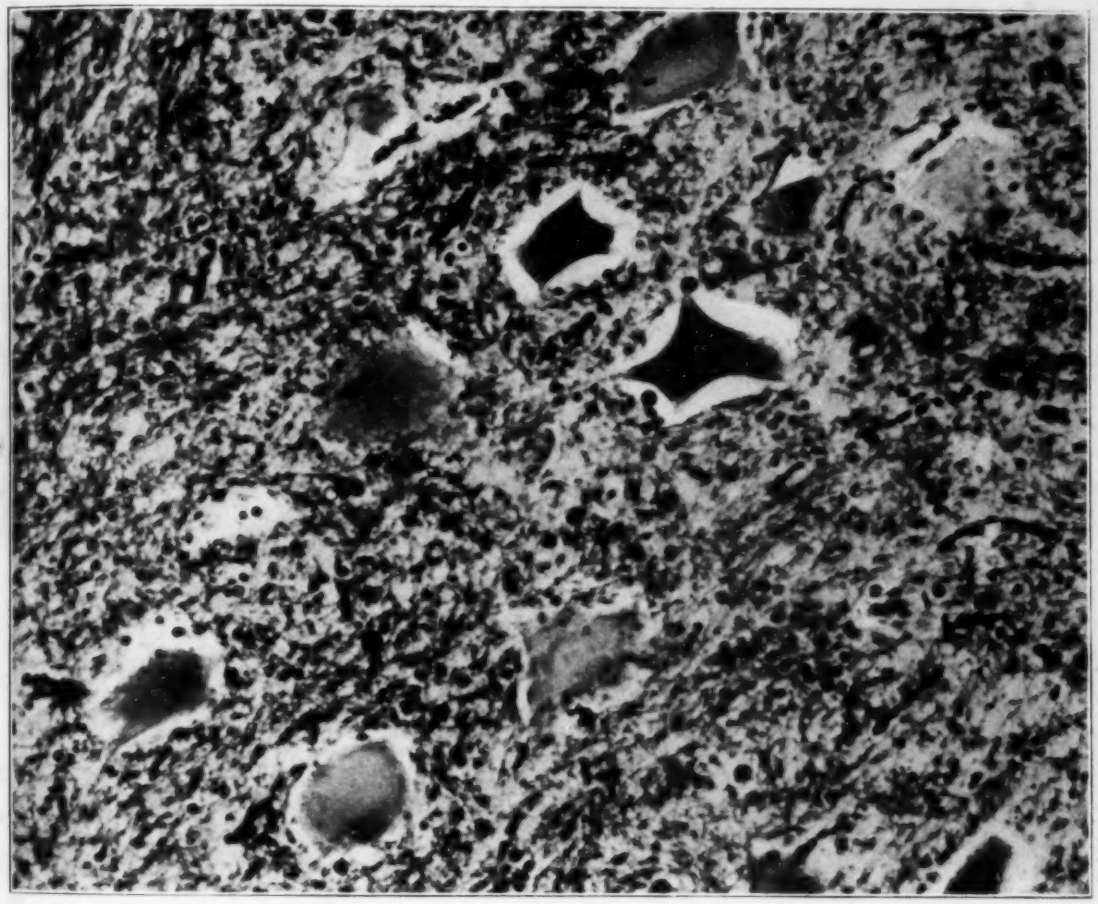

Fig. 4.—Anterior horn cells of the lumbar spinal cord. Note central chromatolysis, swelling, eccentric nuclei and probable cell death. Two of the cells show marked shrinking. Note absence of reactive cells.

in the hypoglossal nucleus and to a more marked degree in the dorsal motor nucleus (fig. 8) and nucleus ambiguus. The dorsal motor nucleus of each side was the site of an unusually marked infiltration with amyloid bodies. The cells of the tractus solitarius and descending root of the trigeminal nerve, aside from a slight cloudiness, showed little or no change. Nowhere throughout the medulla

was there any cellular infiltration of endothelial cells, polymorphonuclear leukocytes or phagocytic glia cells.

Comment.—This case was studied in detail because, of the entire group, it was the only one in which there were no conflicting toxic fac-

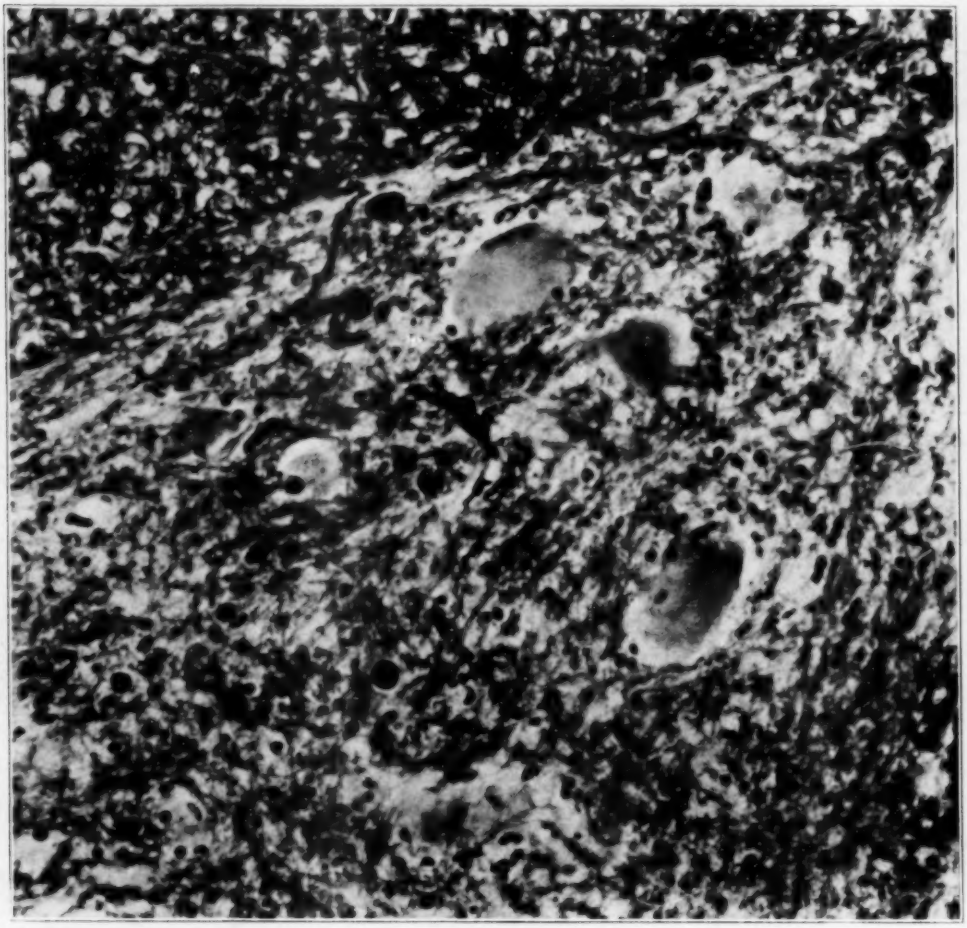

Fig. 5.—Anterior horn cells at the level of the cervical enlargement. Severe chromatolysis, eccentric nuclei and probable cell death are shown.

tors other than the Jamaica ginger extract itself. The patient died of bulbar paralysis after paralysis which involved first the feet and hands and finally the muscles of speech, swallowing and respiration. Autopsy confirmed the absence of other severe pathologic factors. While some slight degree of systemic arteriosclerosis was noted, microscopic examination of the nervous system revealed an absence of arteriosclerosis of

the cerebral vessels (fig. 9). The most striking factor revealed by a study of the material is the presence of marked degenerative changes in the anterior horn cells and in the peripheral nerve fibers without any sort of inflammatory reaction, as shown by the absence of the usual reactive types of cells. Because of this, it can be regarded as demon-

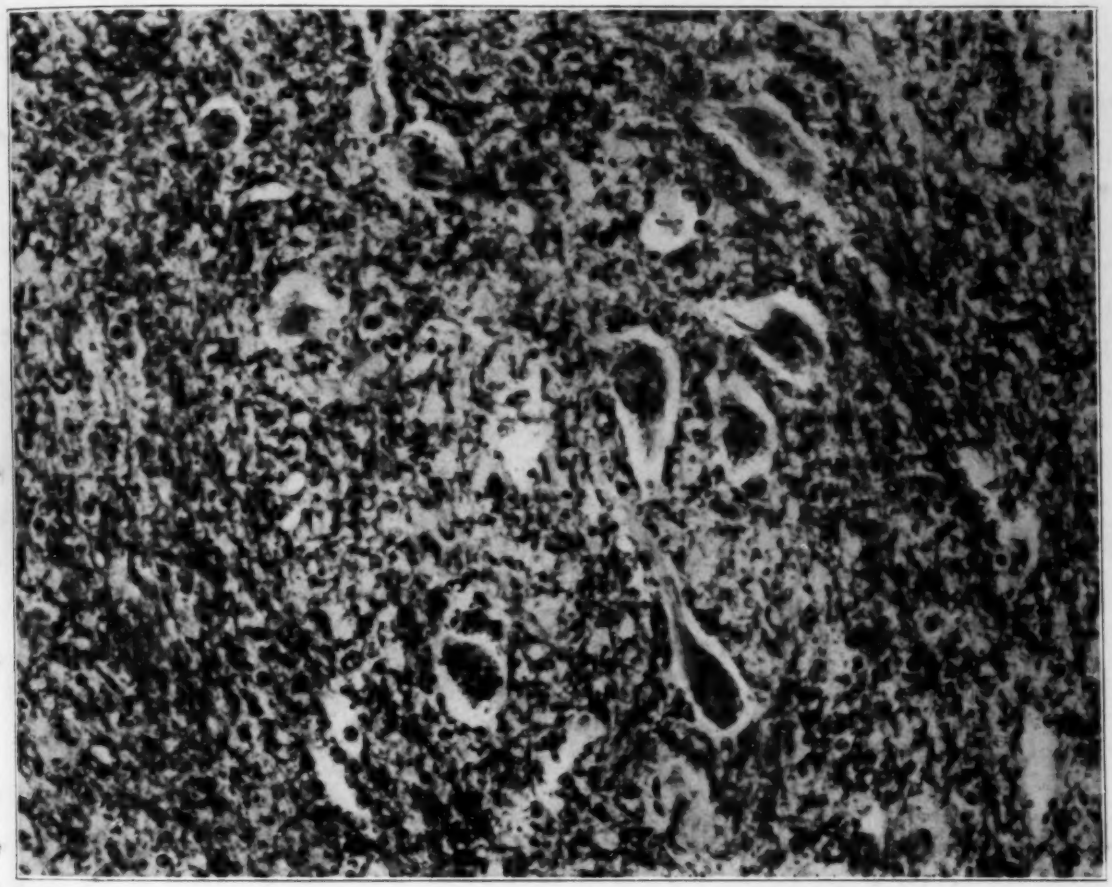

Fig. 6.—Section through the first cervical segment showing a group of normal anterior horn cells. This group apparently escaped injury and form a convenient basis of comparison with cells elsewhere in the spinal cord and medulla.

strated that the toxic agent producing the paralysis was definitely non-infectious.

REPORT OF OTHER CASES

Three other cases which came to autopsy presented areas of the nervous system for study. In every one, autopsy revealed a pathologic factor in addition to Jamaica ginger extract. These cases are regarded

as of value because they present corroborative evidence of the pathologic changes noted in the uncomplicated case 1.

CASE 2.—*History.*—G. B., a man, aged 44, complaining of swollen ankles and inability to walk, entered the Cincinnati General Hospital on March 24, 1930. The patient had been drinking Jamaica ginger extract for about one year.

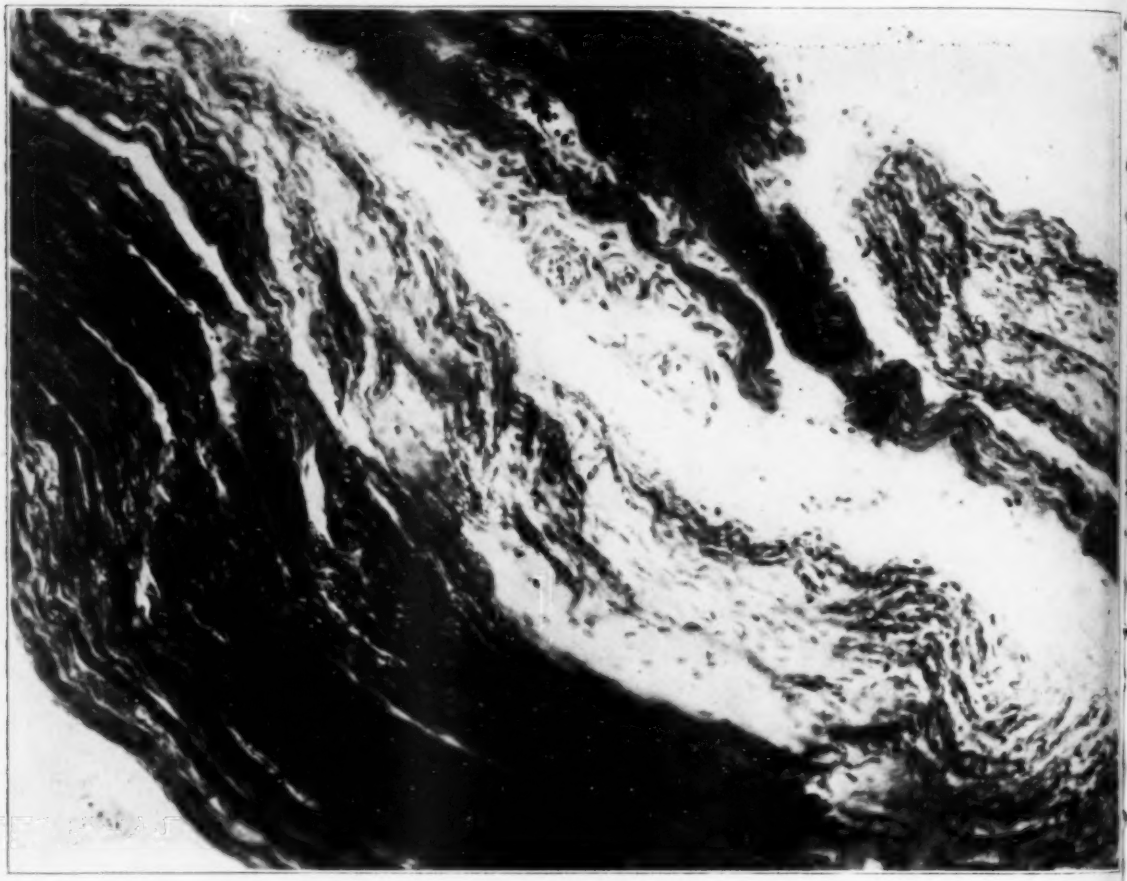

Fig. 7.—Longitudinal section through the root of the vagus nerve showing numerous degenerated myelinated fibers.

Examination.—Respiration was wheezing, and there were many piping râles. There were bilateral foot drop and wrist drop; the hand grip was very weak. The patellar and achilles reflexes were present. There were no pathologic reflexes. The cerebrospinal fluid showed globulin, 1+, a pressure of 15 cm. and a negative Wassermann reaction. The Wassermann reaction of the blood was negative.

Course.—The patient gradually became worse and died of cardiac failure and pulmonary edema.

Autopsy.—The anatomic diagnosis was: marked acute nephritis and nephrosis, acute pyelitis, active chronic fibrous pulmonary tuberculosis, subacute bronchitis and visceral evidences of toxemia. Examination of the brain was made after fixation in formaldehyde. The blood vessels and their immediate branches at the base of the brain showed no striking abnormalities aside from occasional atheromatous plaques. The pia-arachnoid membrane at the base of the brain showed nothing abnormal. The blood vessels over the cortex were moderately congested.

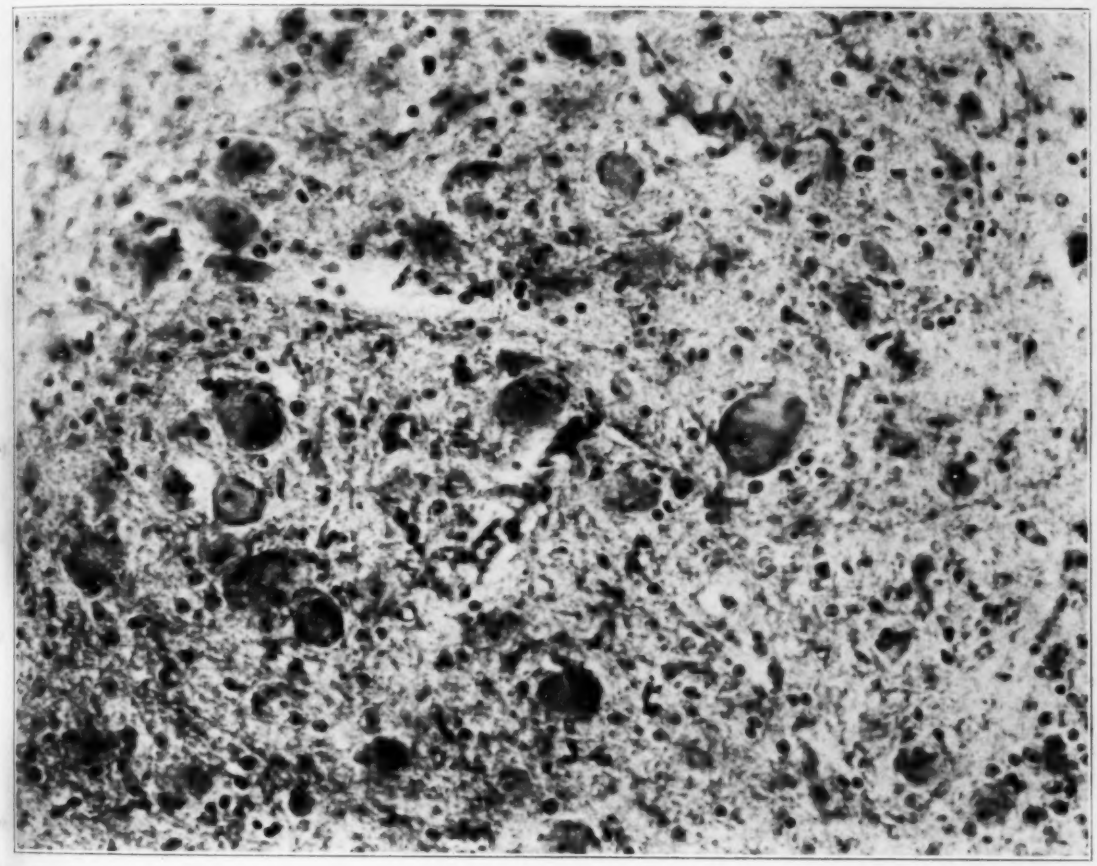

Fig. 8.—Section through dorsal motor nucleus of the vagus showing swelling and chromatolysis of nerve cell bodies.

The convolutions were diminished in size and narrowed and flattened on their surface. The pia-arachnoid over the frontoparietal areas was heavily clouded and thickened. A horizontal section through the brain revealed no striking abnormalities. The pia-arachnoid membrane of the spinal cord showed nothing abnormal. A section through the cervical enlargement showed no striking abnormalities aside from slight prominence of the vascular markings. The following diagnosis was made: pial thickening and clouding; moderate cerebral edema and congestion, and cortical atrophy.

Microscopic sections through the cervical and lumbar enlargement revealed a diffuse infiltration of amyloid bodies, a slightly thickened pia-arachnoid membrane, the fiber tracts intact, occasional degenerative fibers in the anterior roots, numerous swollen anterior horn cells with chromotolysis and eccentric nuclei and many spaces representing areas of destroyed anterior horn cells. There were no proliferative glia cells, polymorphonuclear leukocytes or endothelial leukocytes.

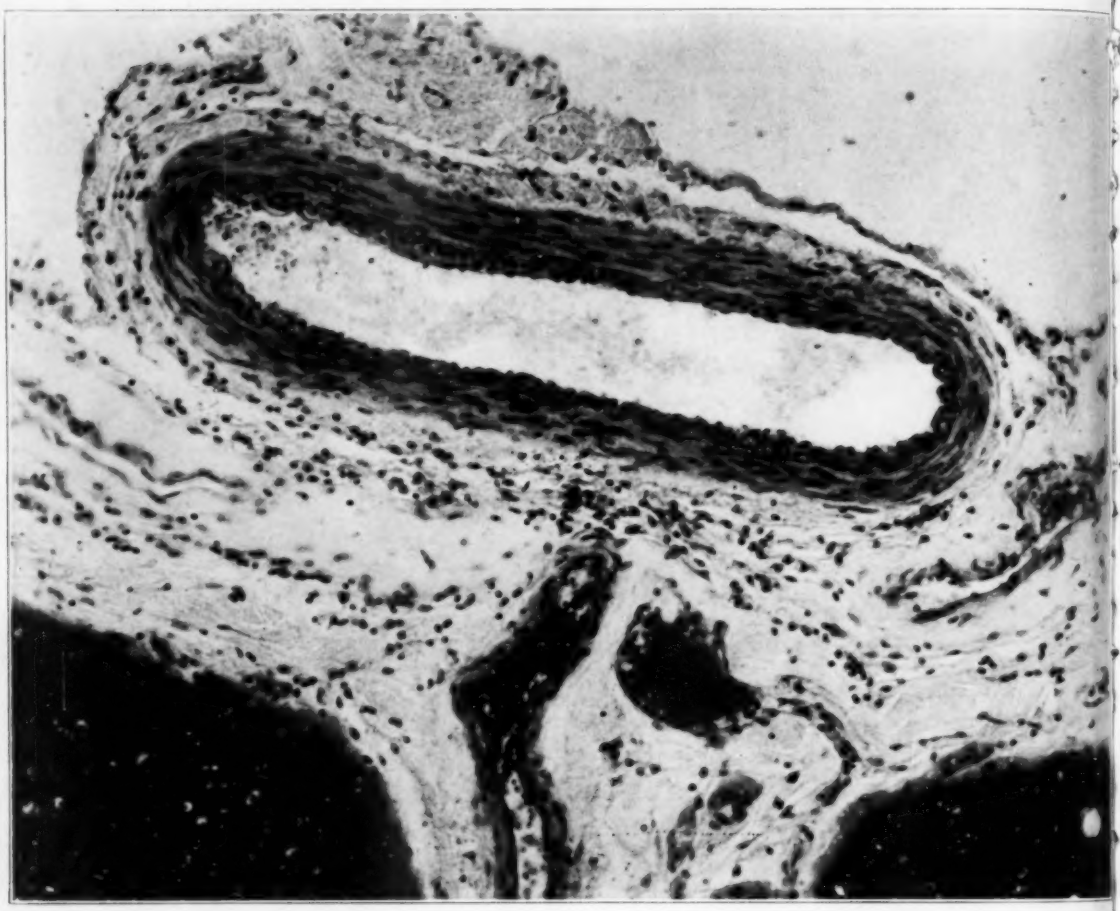

Fig. 9.—Anterior spinal artery in upper cervical cord. Note smooth contour and absence of atheromatous and other pathologic changes. This vessel is typical of the arteries throughout the nervous system.

Comment.—This case presents the clinical history of Jamaica ginger poisoning with the typical manifestations of foot drop and wrist drop. The case was complicated by marked acute nephritis and pyelitis and acute pulmonary tuberculosis.

CASE 3.—*History.*—R. G., a man, aged 45, complaining of pain and weakness in the legs, was admitted to the Cincinnati General Hospital on March 12, 1930. He had been drinking two bottles of Jamaica ginger daily for about a week. He had noticed numbness in both legs, pain and gradual loss of ability to walk.

Examination.—The patient was bedridden. The pupil of the right eye was larger than that of the left; both were contracted and fixed to light. There was a fine tremor of the extended hands. The pharyngeal reflexes were absent. The deep reflexes were hyperactive. The blood pressure was 178 systolic and 104 diastolic. The cerebrospinal fluid gave a strongly positive Wassermann reaction; the gold curve showed precipitation in the dementia paralytica zone; the pressure was 13 cm.; the sugar was 50 mg.; there were 175 cells.

Course.—The patient complained of stricture. The stricture was dilated without bleeding. Twenty-four hours later, the respiration was uremic. A retention catheter was inserted, and a transfusion was given, but the patient died on April 20.

Autopsy.—The anatomic diagnosis was: acute gangrenous cystitis, generalized toxemia, syphilis of the central nervous system, syphilitic aortitis, lobular pneumonia, acute bronchitis and arrested pulmonary tuberculosis. After fixation in formaldehyde, the blood vessels of the brain and their immediate branches at the base of the brain were found to contain atheromatous plaques. The posterior columns presented a grayish, translucent appearance as compared with the rest of the substance of the spinal cord. The following diagnosis was made: moderate cortical atrophy and posterior spinal sclerosis.

Sections through the lumbar, thoracic and cervical cord revealed early sclerosis of the posterior columns, which was more marked in the lumbar area. There were proliferation of glia cells and some perivascular infiltration. The pia-arachnoid membrane was thickened and infiltrated with numerous lymphocytes and mononuclear cells. In addition, there was diffuse infiltration with amyloid bodies. The anterior horn cells showed acute Nissl changes, chromatolysis, swelling and eccentric nuclei.

Comment.—This case of Jamaica ginger poisoning was complicated by toxemia, gangrenous cystitis and syphilitic pathologic changes.

CASE 4.—*History.*—W. J., a man, aged 56, with a history of having taken Jamaica ginger for several years, was admitted to the Cincinnati General Hospital on March 25, 1930, complaining that nine days before admission he had suffered from severe diarrhea which had lasted for five days. There was numbness from the knees downward and in the finger tips.

Examination.—The patient had respiratory difficulty and there were many coarse and fine râles throughout the chest. There were bilateral foot drop and wrist drop. The patellar reflexes were hyperactive; the achilles reflexes were not obtained. There was no response to plantar stimulation. The pupils were dilated and fixed.

A comatose condition developed rapidly, and the patient died one day after admission.

Autopsy.—The anatomic diagnosis was: far advanced chronic glomerulonephritis superimposed on a toxic nephrosis, far advanced pulmonary tuberculosis, compensatory pulmonary emphysema, chronic pleuritis, syphilitic aortitis, acute bronchitis, subacute gastritis, chronic cystitis, dental caries and pyorrhea alveolaris.

After fixation of the brain in formaldehyde, a marked congestion of the blood vessels at the base of the brain was found. The pia-arachnoid membrane was somewhat thickened and cloudy at the base and definitely thickened and cloudy over the cortex. There was a moderate degree of congestion of the veins draining into the superior longitudinal sinus; the convolutions, however, were moderately well rounded. In a horizontal section through the brain, there was evidence of considerable wrinkling, suggestive of dehydration. Sections through representative levels of the spinal cord showed marked reddening of the gray matter. The following diagnosis was made: cerebral congestion, pial thickening and clouding and congestion of the spinal cord, most marked in the cervical region.

Microscopic sections through the cervical and lumbar enlargement of the spinal cord revealed an intact pia-arachnoid membrane, moderate congestion of the blood vessels throughout the spinal cord and diffuse infiltration of amyloid throughout the cord. The anterior horn cells showed marked chromatolysis and eccentric nuclei, occasional shrunken cells and some apparently normal cells. There were no phagocytic cells, no polymorphonuclear leukocytes and no perivascular infiltration. The fiber tracts presented nothing abnormal. There were occasional degenerated fibers in the anterior roots. Sections through the cerebellum revealed Purkinje cells slightly clouded but otherwise intact in number and form. Sections through the various areas of the cortex revealed numerous swollen cortical cells.

Comment.—This case presents the clinical symptoms of poisoning with Jamaica ginger in the presence of far advanced chronic glomerulonephritis.

SUMMARY AND CONCLUSIONS

Four cases of poisoning with commercial extract of Jamaica ginger are reported. One case is reported in detail. This case was uncomplicated by other pathologic factors, and involved death by ascending paralysis and bulbar involvement. The pathologic studies revealed degenerative changes in the radial and anterior tibial nerves, degenerative changes in the anterior roots, marked pathologic changes in the anterior horn cells, characterized by swelling, chromatolysis, eccentric nuclei and death of the cells, and occasionally by shrinking of the cells. Central chromatolysis was the predominating type of cell change. All sections showed infiltration with amyloid bodies. The sensory nuclei were only slightly affected. The fiber tracts of the brain stem and spinal cord remained unaffected. Severe cellular changes, similar to those found in the anterior horn cells, were noted in the hypoglossal nucleus, dorsal motor nucleus of the vagus and nucleus ambiguus. There were no pathologic signs of inflammation, no phagocytic glia cells, lymphocytes, polymorphonuclear leukocytes or endothelial cells.

The pathologic observations suggest a toxin which reaches the peripheral nerves in the extremities through the circulation, but which subsequently travels into the central nervous system along the nerve fibers and anterior roots. Thus, in addition to the central chromatolysis in the motor cells of the anterior horn, associated with involvement of

the peripheral nerve, diffuse chromatolysis and death of the cells also occur. The toxin, in severe cases, may then make its way to the medulla, where involvement of the vagal nuclei produces death by bulbar paralysis. There is an obvious tendency of the toxin to exert its pathologic effect most severely on motor cells and fibers.

The pathologic observations can hardly be regarded as characteristic of any one toxic agent. They serve to eliminate, however, all possibility of the "epidemic" being infectious in origin.

CONGENITAL CEREBRAL CYSTS OF THE CAVUM SEPTI PELLUCIDI (FIFTH VENTRICLE) AND CAVUM VERGAE (SIXTH VENTRICLE)

DIAGNOSIS AND TREATMENT *

WALTER E. DANDY, M.D.
BALTIMORE

In the midline of the brain and within the confines of the corpus callosum either or both of the cavum septi pellucidi and cavum vergae are not infrequently found. Neither cavity has excited much interest either anatomically or clinically. The two cases here reported are, I believe, the first instances in which a diagnosis of these spaces, dilated in abnormal degree, has been made during life, the first in which the lesion has been found at or treated by operation and the first in which clinical symptoms are shown to be related to the lesions. But there is reason to believe that these cavities may not be uncommon and may not be unimportant in clinical neurology. Moreover, the diagnosis of the lesion is easy and unequivocal. The operative treatment is not difficult, though not unassociated with great danger if certain well defined precautions are not recognized. A permanent cure of the lesion can probably be expected.

In most, but not all, textbooks of anatomy there is a brief but accurate description of these two cavities. The nomenclature, however, is not uniform. For example, the cavum septi pellucidi is perhaps better known as the fifth ventricle, and the cavum vergae is called Verga's ventricle, the sixth ventricle, the ventricle of Strambio, ventriculus fornicis, ventriculus triangularis and the canal aqueduc.

The cavum septi pellucidi, the much better recognized of the two, has been known at least since the time of Sylvius and probably much longer. The cavum vergae is named for Andrea Verga, an Italian anatomist, whose observations were reported in a brief note (1851) written in response to the report by a fellow anatomist, Ferrario, of a necropsy in a case showing the cavity that bears Verga's name. Apparently, Verga's claim to priority is based on his anatomic notes rather

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* Read at the Fifty-Sixth Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 9, 1930.

* From the Johns Hopkins University and Hospital.

than on priority of publication. The following excerpts from Verga's [1] letter to Ferrario show the exact status of Verga's claim to priority, which according to present tenets based on prior publication and not the date of actual observations, should go to Ferrario:

Your gracious letter, directed to our esteemed teacher, C. Panizza, and inserted in the January number of the *Gazetta medica*, comes at a most opportune time. Your interesting publication of "an unusual finding in the brain of a man who had never presented signs of mental alienation" made me resolve to publish an old note of mine about a new cerebral ventricle which receives from your observation a great impetus and a great importance. And to you—an unknowing illustrator of my discovery (let me call it mine until proven otherwise)—I first communicate this note. When I was assistant to the chair of anatomy in Pavia, examining one day the brain of a child dead of hydrocephalus, I was surprised to find . . . this cavity. I repeated the observation upon other brains . . . and always found the said cavity more or less evident, . . . but in this age of the microscope, I made nothing of the observation, for I thought there was nothing that could be seen by the naked eye that was not already known to all. Perhaps with time I might have forgotten it, had not your observation come to revive the memory of it. Now that you, learned in so many things, and especially anatomy, appear not to know that others had spoken of the cavity, I suspect that none really has ever described it.

Verga then proceeded to describe the cavity and gave directions how to find it:

Not knowing that this ventricle was something really new . . . I refrained from giving it a name. It is, therefore, an innominate ventricle. But a numerical number could be given to it—sixth ventricle—or from its shape it might be called the triangular ventricle, or from its relations, the ventricle of the vault.

He then discussed Ferrario's contention that the ventricle is an abnormality and not a uniform anatomic observation.

Before finishing I should like to remove a doubt which, it seems to me, exists in your mind. If this cavity, you say, really exists and is a normal structure it would be found in all cadavers. I have found practically the same thing in all children and neither did I have to work hard nor employ artifices of any sort. I must, therefore, conclude that it is a natural and normal finding like that of the septum pellucidum; but whether because of age or other circumstances, which are beside the question here, it is very often perfectly obliterated in the adult.

Concerning his claim to priority, he added in conclusion:

The most serious objection that can be made to my findings is, therefore, that which I myself made long ago, and because of which I delayed so in publishing it. In truth, at this very moment, I am afraid of appearing guilty of a scientific

1. Verga: Dell' apparato ventricolare del setto lucido e della volta a tre pilastri, Mm. r. ist. lomb. di sc., Aug. 2, 1855; Gior. r. Istituto lomb. di sc., nos. 43 and 44, p. 89; Sul ventriculo della volta a tre pilastri, Gazz. med. lomb., no. 7, July 7, 1851.

theft and of having to suffer the brutal consequences. But I declare that I have never had such habits and I have done all this prattling only to explain the unusual lesion which you have observed and reconnected.

In the same publication, Verga mentioned the comment of Tenchini [2] that the ventricle was discovered by Verga and should be called Verga's ventricle.

POSITION AND BOUNDARIES OF THE CAVITIES

The corpus callosum defines the anterior, superior and posterior limits of the two cavities which, when not continuous, are separated from each other by the anterior limit of the fornix as it courses obliquely backward and upward from the anterior commissure to the body of the corpus callosum (fig. 2). Being of congenital origin, the two cavities are doubtless dependent on the development of the corpus callosum and the fornix. They may coexist and be isolated from each other when the fornix is intact (figs. 2 and 3); they may coexist and be in communication through a defect in the fornix, or they may form a single large cavity when the fornix is not attached to the corpus callosum, as in case 1 of this report (figs. 4, 5 and 6). The cavum septi pellucidi is frequently present when the cavum vergae is absent, and Verga's cavity may be present when the cavum septi pellucidi is absent. Kauffmann stated that the cavum vergae may exist on one side of the midline and be absent on the other.

The cavum septi pellucidi has the following boundaries: anteriorly, the genu of the corpus callosum; superiorly, the body of the corpus callosum; posteriorly, the anterior limb and pillars of the fornix; inferiorly, the rostrum of the corpus callosum and the anterior commissure; laterally, the layers of the septum pellucidum. Viewed laterally, the cavum septi pellucidi is roughly triangular with the base at the corpus callosum. Viewed in cross-section the cavity is also triangular with the base at the corpus callosum.

The cavum vergae has the following boundaries: anteriorly, the anterior limb of the fornix; superiorly, the body of the corpus callosum; posteriorly, the splenium of the corpus callosum; inferiorly, the psalterium (lyra davidis) and hippocampal commissure, the fibers of which bridge the space between the diverging posterior pillars of the fornix. This cavity is also triangular when viewed from the side. The cavum vergae flares out laterally on both sides with the curve of the fornix and pushes under the lateral ventricles at its extreme lateral extensions. Although the floor of Verga's cavity rests on the tela choroidea which contains the choroid plexus, evidence of choroid plexus has not been found within, but there is no available report of careful studies of the entire wall.

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2. Tenchini, L.: Contributo alla storia dei progressi dell' anatomia e della fisiologia del cervello, Naples, 1880, p. 174.

COMMUNICATIONS WITH THE VENTRICULAR SYSTEM

In most adult brains both spaces are absent or are at most potential, but in every 100 necropsies actual cavities of varying size will be seen. Neither cavity can be regarded as part of the great ventricular system

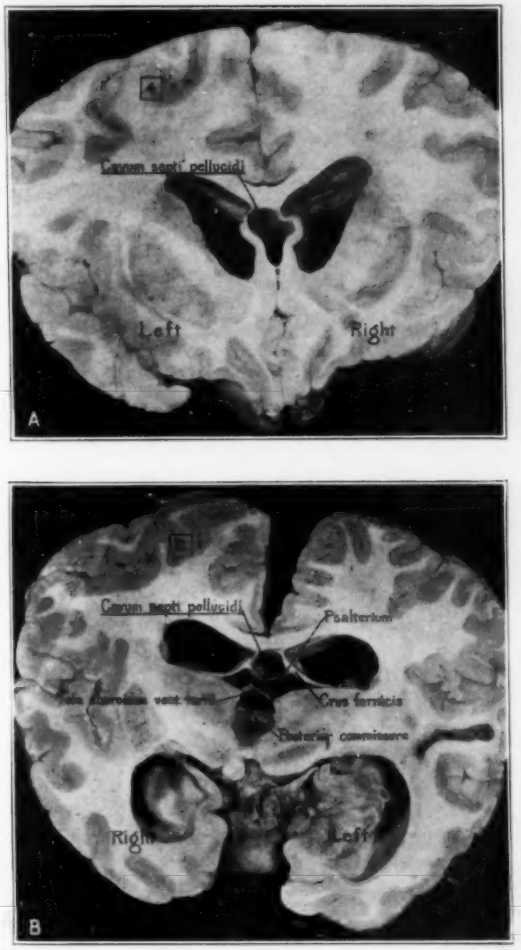

Fig. 1.—*A*, characteristic cavum septi pellucidi; note the varying thickness of the walls of the septum. *B*, cavum septi pellucidi at the junction of the cavum vergae. The artificial space between the psalterium and the tela choroidea may be mistaken for the cavum vergae. Note the shreds of tissue between the structures indicating that it is not a natural cavity.

in which cerebrospinal fluid forms and through which it circulates. They contain no gross evidence of choroid plexus, and their development in the embryo is entirely different; any connection with the

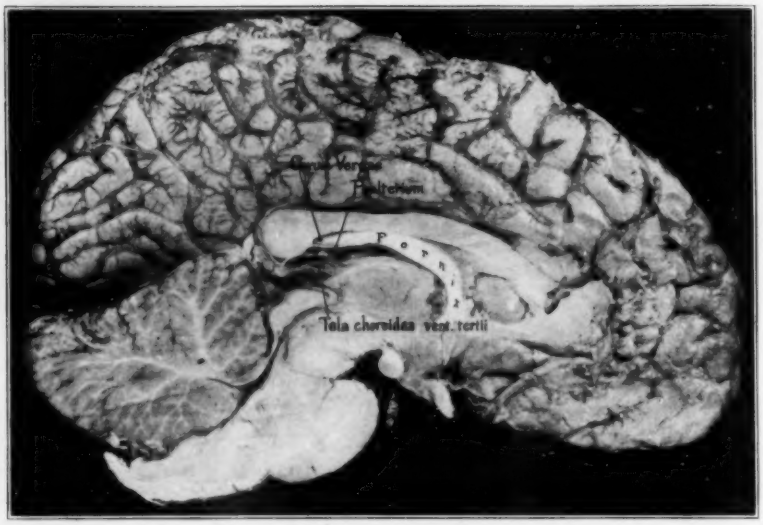

Fig. 2.—Small slitlike cavum vergae.

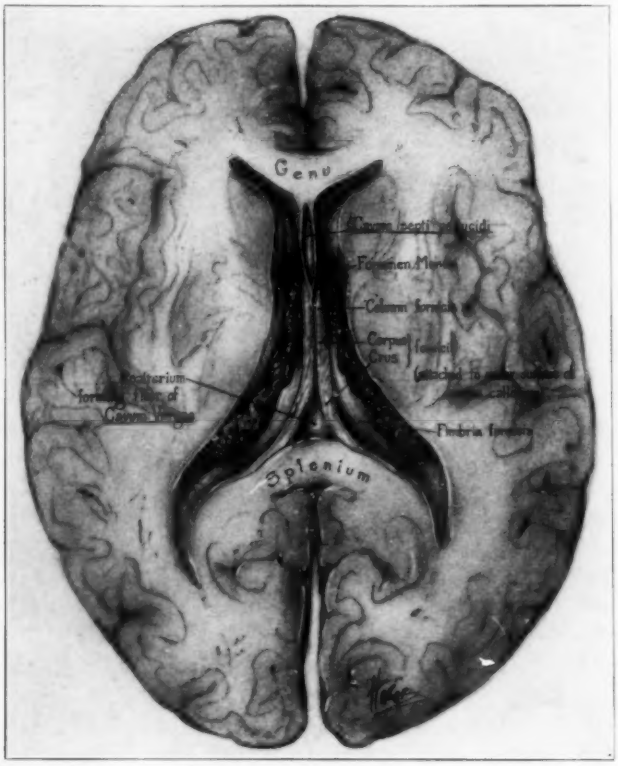

Fig. 3.—Cavum septi pellucidi and cavum vergae, each independent.

ventricular system is purely accidental and is due to contiguity. It is therefore inadvisable to retain the term "ventricle" with the numerical designations. In most necropsy specimens both of these cavities, when of unusual size, are found to communicate with the ventricular system by one or even more openings of varying size (figs. 5, 6 and 7). The openings are into the third and lateral ventricles. In some textbooks of anatomy, names have been awarded to these openings, i. e., the foramen or valve of Vieussens, the foramen of Mihalkowski, etc. Verga stated

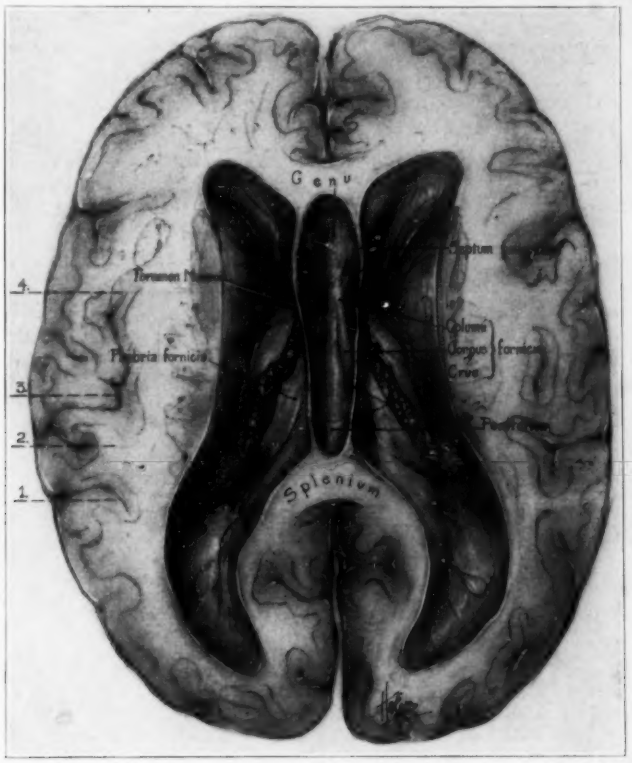

Fig. 4.—Large cavity formed by union of the cavum septi pellucidi and the cavum vergae.

that Petit (1710), and in more recent times Wenzel, Tiedemann, Meckel and Valenin, considered the openings between the cavity of the septum pellucidum and the ventricular system to be normal. Vicq d'Azyr and Santorini thought that they were artificial, a view which Verga also held.

That these openings are artificial and not preformed like the interventricular foramina is evident by their inconstant position, size and number (figs. 5, 6 and 7), and especially by the fact that their borders

are ragged and uneven, shreds of tissue usually hanging therefrom. The location of the openings is doubtless dependent on the points of greatest thinness of the walls (fig. 1 *A*).

Verga stated that the cavity of the septum pellucidum and the cavity that bears his name are found only in the human species.

CASES COLLECTED FROM THE LITERATURE

Verga reported five cases in which this cavity was found at necropsy. All were from psychopathic wards, the patients showing varying grades

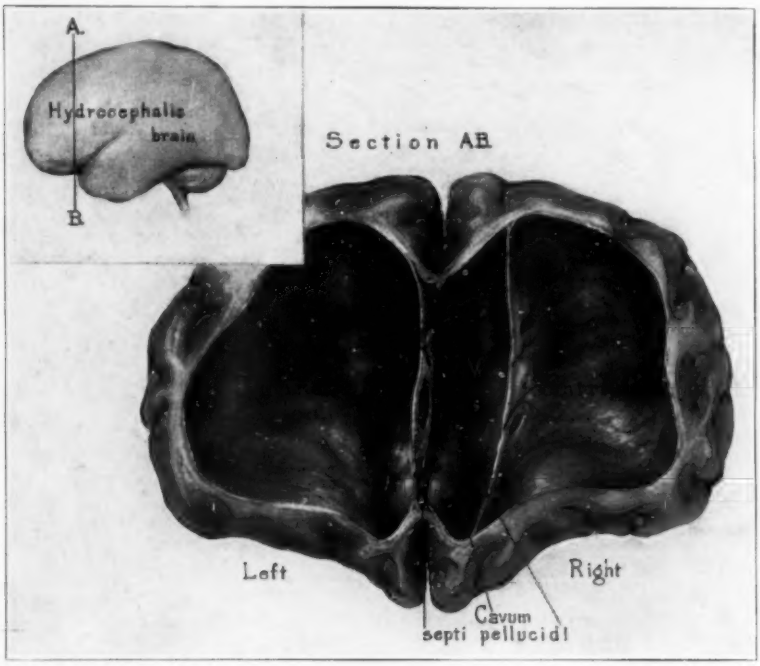

Fig. 5.—A hydrocephalic brain showing a congenital malformation. The cavum septi pellucidi is formed by two distinct layers, one attached far to the right of the midline. There are numerous artificial openings between this cavity and the lateral ventricles.

of mental disturbance. There was nothing characteristic in the assembly of symptoms. In every instance the cavity was small. He also referred to a specimen shown by his friend, Dr. Sangalli. The cyst was as large as an almond. The patient was a girl, aged 17, afflicted with epilepsy. He also noted that Ferrario's case, previously mentioned, was the best one that he had seen. Other cases were shown to him by his confrères, Palermo, Dubini, Biffi and De-Vincenti (1882), Inzani di Parma, Strambio and Tenchini. He quoted Tenchini as saying that the length

may be as much as 13 cm.; this extreme length probably includes the cavity of the septum pellucidum in addition to the cavum vergae. Tenchini also found the cavity in 4 per cent of males and 9 per cent of females, and constantly in fetuses and the new-born.

It will be noted that all of the aforementioned cases were reported by Verga from the observations of his own acquaintances or at least from a restricted territory. There is every reason to believe, therefore, that the occurrence of the cavity is fairly frequent.

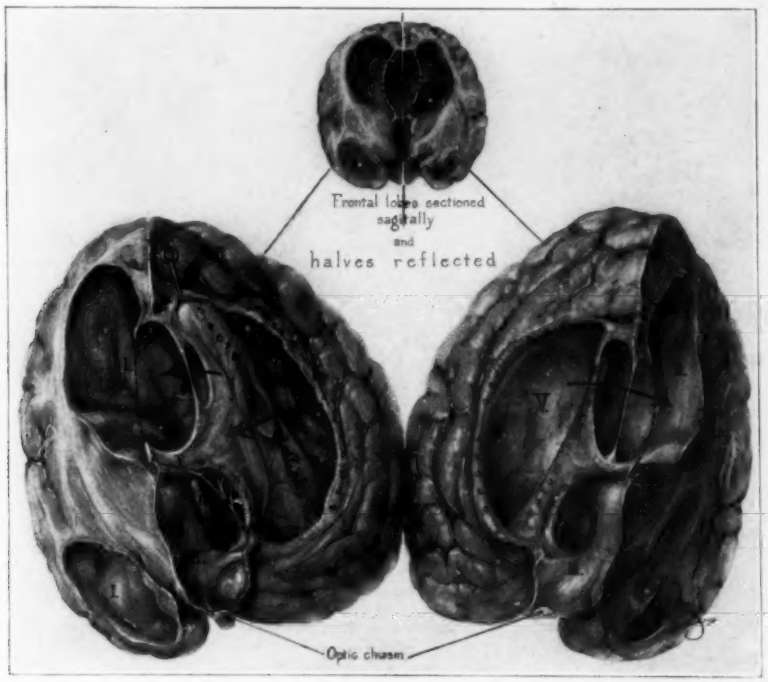

Fig. 6.—Another hydrocephalic brain with huge lateral ventricles, and the cavum septi pellucidi proportionately as large.

Examples of a dilated cavum septi pellucidi are probably even more common, though they are not easily accessible in the literature and so far as I know no effort has been made to assemble them. Kauffmann reported a necropsy showing both cavities moderately distended.

WHERE DOES THE INTRACYSTIC FLUID ARISE?

In their textbooks, Dejerine and Poirier and Charpy stated that microscopic examinations of the walls of the cavum septi lucidi have been made, and that the epithelial cells of the ependyma and choroid plexus are uniformly absent. The evidence, however, is not sufficient to

accept without further proof. One now knows that cerebrospinal fluid forms from choroid plexus and also that these cavities lie on the tela choroidea (though normally separated from it) which forms the roof of the third ventricle. Since these cavities in dilated form are uncommon, it is open to question, until disproved by more extensive microscopic studies, whether minute remnants of such cells may not be included occasionally.

Testut and Reichert have explained the existence of fluid in these cavities on the basis of transudation from the lateral ventricle, but proof of this theory is lacking. It seems much more probable that the fluid

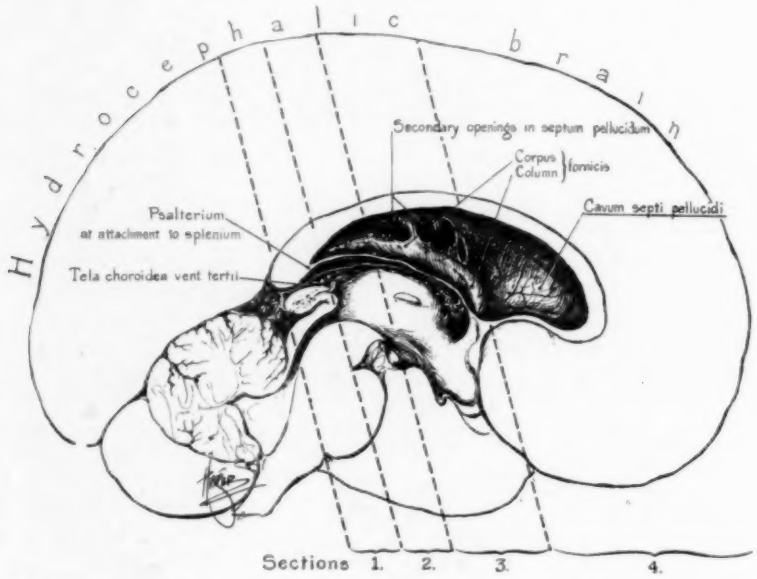

Fig. 7.—Sketch of another hydrocephalic brain showing a large combined cavum septi pellucidi and cavum vergae. There are artificial openings between this cavity and the lateral ventricles, the position varying markedly from that in the two preceding cases. The presence of either or both of these cavities is not an uncommon observation in hydrocephalic brains.

originates within the cavity, though from a source unknown. There cannot be sufficient evidence to eliminate this source until the entire ventricular wall has been subjected to microscopic study. Certainly in my two cases the walls of the cavity were far thicker than the normal thin transparent septum pellucidum, and I believe this to be an important reason for the isolation and growth of the "cyst," at least until by pressure absorption, erosion of the wall results at its thinnest point. The source of all fluids is not known, but there must be some unusual anatomic basis for the occasional unusual cysts that develop.

REPORT OF CASES

CASE 1.—*History.*—A sparsely nourished woman, aged 50, was referred by Dr. I. Abrahamson of New York with a probable diagnosis of tumor of the brain. Eleven years before, she began to have severe, recurring, generalized headaches, which, however, disappeared five years before the present examination and did not recur. Nine years before, she had a period of unconsciousness which lasted for only a few minutes. There were no later spells of this kind and no convulsions. Three years before, her memory began to fail. She had difficulty in understanding, and she did things incorrectly. She became slovenly in dress and personal habits. Two years before, periods of vomiting began, and in a short time she lost 40 pounds (18.1 Kg.) in weight. No satisfactory explanation could

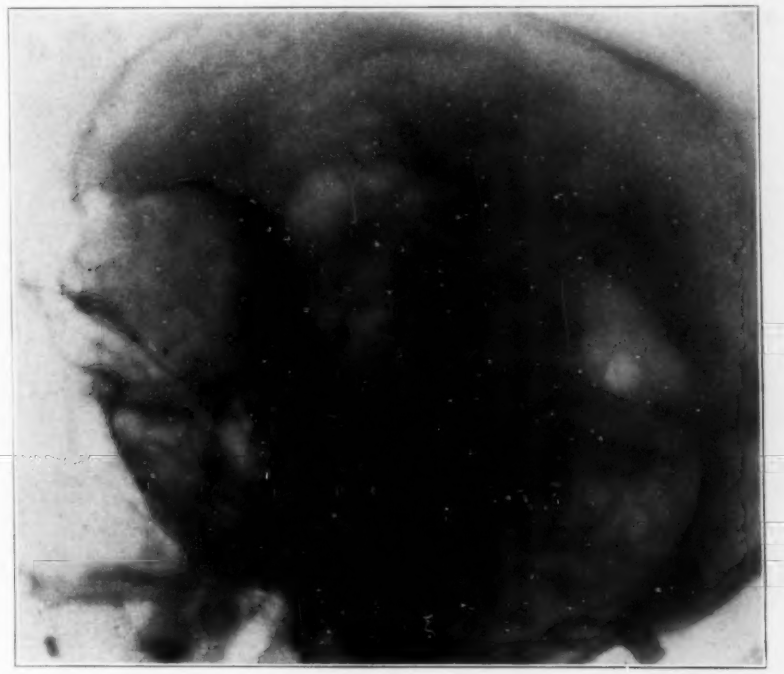

Fig. 8 (case 1).—Lateral ventriculogram of patient with large cavum septi pellucidi and cavum vergae forming a single large cavity.

be found for the vomiting, which later disappeared. Pain in the right arm began about two years before and had since been present at times; for this also no cause was found. Two months before, she was struck by an automobile, but sustained only bruises and was unconscious for only a few minutes. Soon thereafter she had difficulty in naming things correctly. She would put a shoe on the wrong foot. Her memory steadily became worse.

Examination.—The patient was much under weight. She was disoriented as to time, place and person. She thought that she was in a New York hotel. However, she talked fairly rationally at times. She said that her memory was poor, but that this was due to nervousness. She obeyed many commands cor-

rectly, but it was usually difficult for her to understand even simple statements. There was no difficulty in articulation. She could not name simple objects; for example, when asked to name a pencil she described its use. After some groping she frequently found the correct word. At times her speech was fairly coherent and intelligent, although she quickly wandered off the subject and frequently repeated. Cooperation was poor because of lack of understanding.

The neurologic signs were suprisingly few. There was some unsteadiness of gait. The Romberg test was positive, with falling consistently backward and not to either side. There was no ataxia or nystagmus. A positive Babinski sign on the right and hyperactive knee jerks, greater on the right, were the only other positive observations. There was no papilledema. Roentgen examination of the head gave entirely negative results. The blood pressure was 160 systolic and 100 diastolic; the pulse rate was from 70 to 80. The Wassermann reaction of the blood was negative. Ventricular fluid obtained at the time of operation was normal in every respect; there were 3 cells, no globulin and a negative Wassermann reaction.

Impression.—It was my belief that the patient had a tumor of the brain, probably situated deeply in the left hemisphere. The reasons for this diagnosis were the long period of headaches, the mental changes and the positive Babinski sign on the right side. What type of tumor could cause five years of headache and then freedom from headaches for five years was difficult to imagine. An angioma seemed the most likely possibility.

Ventriculography.—On Jan. 13, 1928, a ventricular puncture was made in the posterior horn of the lateral ventricle. The fluid was under no apparent increased pressure. Twenty cubic centimeters of fluid was removed, and the same amount of air was injected. The roentgenographic observations were as follows: In the anteroposterior view the lateral ventricles (in cross-section) were separated about 3 cm. instead of being in apposition (fig. 9). Moreover, the mesial margin of each ventricle was indented; i. e., there was a filling defect in each ventricle. The filling defect was much greater in the left than in the right ventricle. The third ventricle was upright but shortened in the vertical direction and wider than normal. The lateral ventricles were larger than normal; i. e., there was a low grade of hydrocephalus. The body of the lateral ventricle was smaller than the anterior and posterior parts of the ventricle (fig. 8). The diagnosis made on the basis of the ventriculograms was tumor in the region of the corpus callosum.

Operation.—On January 13, a small bone flap was turned down on the left side of the head just in front of the line of the rolandic area. My plan was to slide down the falx and determine the character of the tumor. If it proved to be inoperable, the small bone flap would doubtless be adequate. If there were possibilities of removing the tumor, extension of the bony defect could be made in the desired direction. Three veins were tied in the crossing from the hemisphere to the longitudinal sinus; they were doubly ligated with silk and divided. The anterior horn of the left lateral ventricle was tapped to give additional room. The mesial surface of the hemisphere was then easily retracted and the corpus callosum quickly brought into view and divided longitudinally in its anterior half. This brought me into a large smoothly lined cavity from which a clear fluid escaped. The exact color of the fluid was masked by a little oozing. The cavity looked like a lateral ventricle, but as there was no choroid plexus it was concluded that it must be a cyst. It was of tremendous size, especially in the anteroposterior direction. It extended in the midline to the anterior limit of the corpus callosum and posteriorly further than I could see or reach with an instrument,

even after the corpus callosum had been divided almost as far back as the great vein of Galen. Anteriorly, the cyst was perhaps 3 cm. wide, and a smooth rounded bulge could be seen running into it longitudinally on each side. Puncture with a small aspirating needle yielded cerebrospinal fluid; they were the lateral ventricles. Posteriorly, the cyst was much wider, certainly not less than 6 or 7 cm., and on each side there was a posterior extension which went far back and under the lateral ventricle. The tip could not be seen. The posterior extension was doubtless limited by the splenium of the corpus callosum which it seemed to parallel (fig. 10 and 12).

The nature of this tremendous cyst, which I estimated to be not less than 12 cm. long, was not clear at the time. The possibility of an enlarged cavum septi pellucidi first occurred to me, but this cavity extended much farther backward and laterally than was possible for a fifth ventricle. I clung to the view that it was probably a cyst, and that there must therefore be an underlying papil-

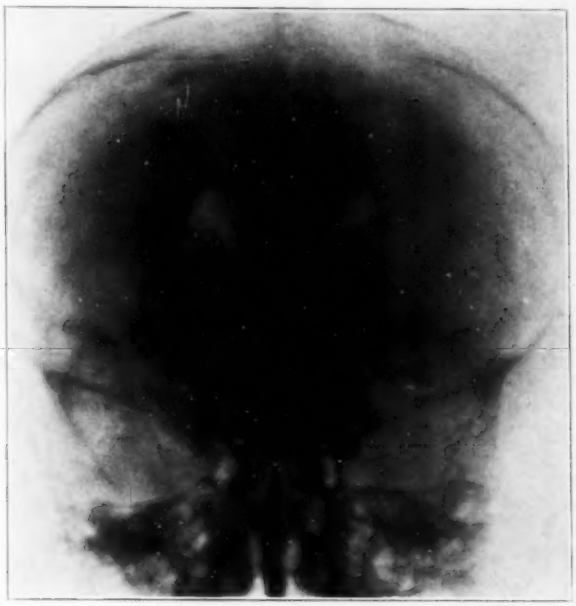

Fig. 9 (case 1).—Anteroposterior ventriculogram in same case showing the characteristic separation of the anterior horns and the base of the lateral ventricles.

loma somewhere in the wall to cause the cyst. It was this erroneous assumption that led me to attempt a careful and thorough inspection of the whole cavity. For this purpose the corpus callosum was split to the great vein of Galen. (On several occasions when tumors of the third ventricle were removed, I had divided the body of the corpus callosum in its entire anteroposterior extent without any untoward results.) With the exception of the posterior lateral extensions of the cavity under the lateral ventricle, the entire cavity could be explored. It was everywhere smooth and glistening, but without choroid plexus and without the larger vessels which line the lateral ventricles. At no point was there an opening into either lateral ventricle or the third ventricle. There was no evidence of a tumor at any part of the wall. The conclusion was therefore forced on me that

it must be, after all, an enlarged cavum septi pellucidi. If this was true, or even if a small tumor existed, opening of the cavity into the lateral ventricle should produce a cure. Openings were then made into each lateral ventricle with forceps. On puncturing the left ventricle some bleeding followed, presumably from the choroid plexus. Both ventricles filled with blood, which, however, was removed. Before closure was begun, all bleeding had stopped, and there was no particular apprehension of this postoperative complication. A decompression was performed as an extra precaution.

Subsequent Course.—The patient died three weeks later, without regaining consciousness. There was never any fulness of the area of decompression. Loss of consciousness was due to thrombosis of the left anterior cerebral artery, which was doubtless injured during the exposure. This is one of the striking

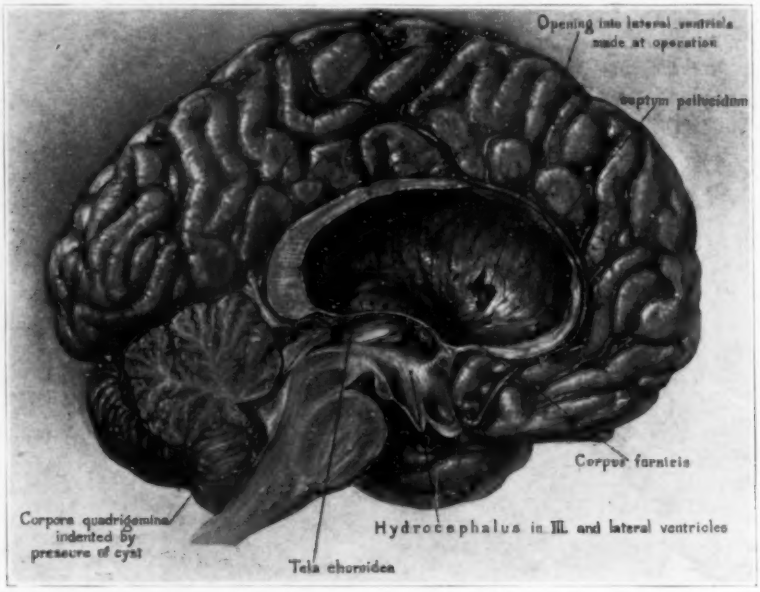

Fig. 10 (case 1).—Midsagittal view of necropsy specimen showing large cyst formed by union of the cavum septi pellucidi and the cavum vergae. The only opening in the lateral wall on either side is the one produced at operation. The absence of communication is also shown by the absence of air between the two ventricles shown in figure 9.

cases from which it has been determined that consciousness lies at some point in the left cerebral hemisphere and within the blood supply of that part of the middle cerebral artery lying above the anterior third of the corpus callosum.

Necropsy showed no other gross changes in the brain.

Case 2.—*History.*—A boy, aged 4½ years, referred by the department of pediatrics, had been born slightly prematurely at 8½ months and his weight at birth was only 2½ pounds (1.12 Kg.). He was blue for one day, after which his color was normal. At birth the head seemed to be abnormally large. The first tooth appeared at 1 year; he sat alone at 1 year, walked at 2 years and talked at 2 years. The mother did not think that the head had

grown faster than the body. The father said that the patient had always been mentally backward. Until six hours before admission to the hospital, the patient had been physically well though mentally backward. He had gone to bed as well as usual. At 3 a. m., he awoke screaming. He did not complain of any pain, but had an attack of vomiting (this was said to have been projectile). He was soon quieted, but awoke crying several more times. During the night he voided in bed, an unusual incident for him. The following morning he was drowsy. Prior to this time he had frequently had nocturnal crying spells, but had always seemed normal in the morning.

Because of the marked drowsiness he was brought to the hospital. At that time he was comatose and was having repeated left-sided convulsions, involving the face, arm and leg.

Examination.—The patient was comatose. He had a very large head, measuring 56 cm. in circumference; it was definitely out of proportion to the rest of his

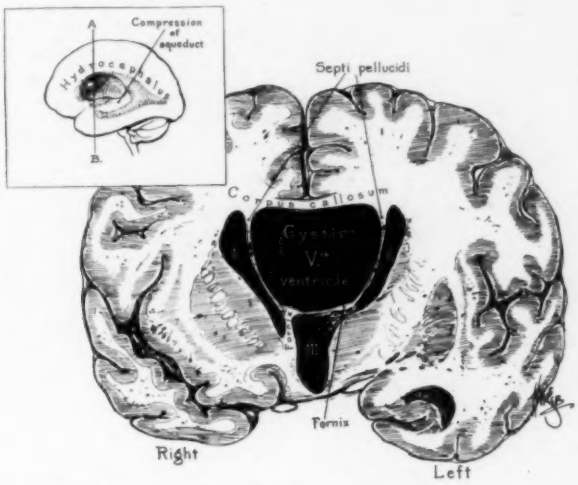

Fig. 11.—Position of congenital cyst in relationship to the ventricular system.

body. Left-sided convulsions were frequent, but without the jacksonian "march." In fact, the convulsions were of a variable character. Occasionally both legs and one (either) arm would jerk. There was at times flaccid paralysis of the entire left side; at other times only the left arm was said to have been paralyzed. The ankle jerk was absent on the left side; the biceps and triceps reflexes were equally active on both sides. A positive Babinski sign was present on both sides, but was greater on the right. Rectal and vesical sphincter control was lost. The abdominal and cremasteric reflexes were not obtainable. The eyegrounds were normal. The temperature was 103.5 F. Aside from a large head the roentgen examination gave negative results.

Course.—On the afternoon of the day of admission, he was sitting up and again moving the left side apparently normally. The spinal fluid was normal. The Wassermann reaction of the blood and spinal fluid was normal.

Dr. Hodge, the resident on pediatrics, made the following note. "The striking things about the history and physical examination are: (1) questionable enlarge-

ment of the head at birth and definite enlargement at present; (2) marked mental retardation which has always been present; (3) some dilatation of the veins over the forehead and otherwise negative physical examination. The paralysis has now entirely cleared. He now runs about aimlessly, laughing in a silly, meaningless fashion. He can say few words but cannot repeat many simple words and does not know his name."

Clinical Diagnosis.—Because of the unilateral convulsions, followed by paralysis of the left side of the body, I thought of a cerebral tumor of congenital origin, perhaps an angioma. In support of this diagnosis was a rather large head. Ventriculography was suggested and was employed a few days later.

Ventriculography.—The right ventricle was reached at the posterior horn and found to be large. Thirty cubic centimeters of fluid was aspirated and an equal amount of air injected.

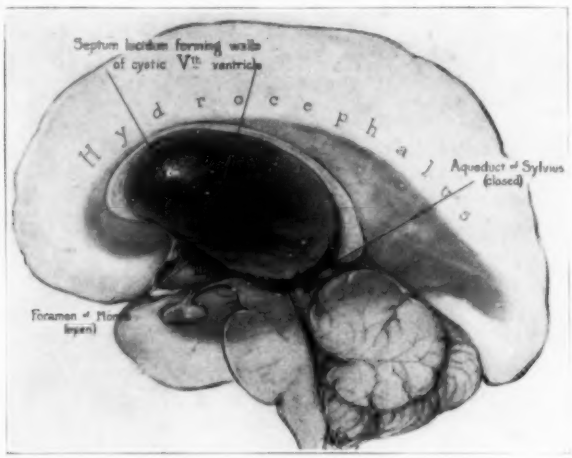

Fig. 12.—Midsagittal view of brain showing relationship to the ventricular system in this place. The mild grade of hydrocephalus which resulted in this case was probably by the pressure on the aqueduct of Sylvius.

A positive diagnosis of an enlarged fifth ventricle was made on the basis of the precisely similar ventriculographic changes in the preceding case. There seemed no other possible lesion that could cause this ventricular deformation at this early period of life.

In the anteroposterior view the two lateral ventricles (in cross-section) were about 2 cm. apart instead of being in apposition. Moreover, the mesial aspect of each ventricle was concave, and the right ventricle was rounded on its superior aspect, a positive sign of a hydrocephalic ventricle (fig. 13).

In the lateral view the posterior horn and a small adjoining part of the body of the ventricle were much enlarged, but anterior to this point the body of each ventricle was reduced in size (fig. 17). At the tip of the anterior horn the ventricles again became larger, the right more so than the left.

From these observations it was concluded that there must be a space-occupying lesion (i. e., a tumor) of some kind which separated the lateral ventricles, indented them locally and caused a mild grade of hydrocephalus by blocking the aqueduct of Sylvius.

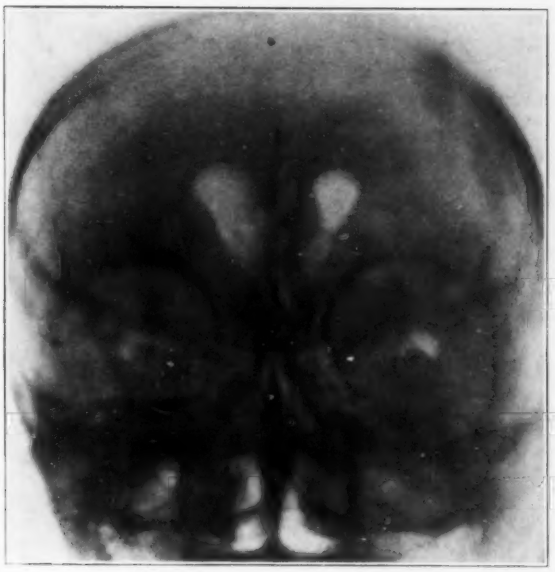

Fig. 13 (case 2).—Anteroposterior ventriculogram showing the same condition as in case 1. The anterior horns of the ventricles are separated and the left anterior horn shows a filling defect from the cyst of the cavum septi pellucidi.

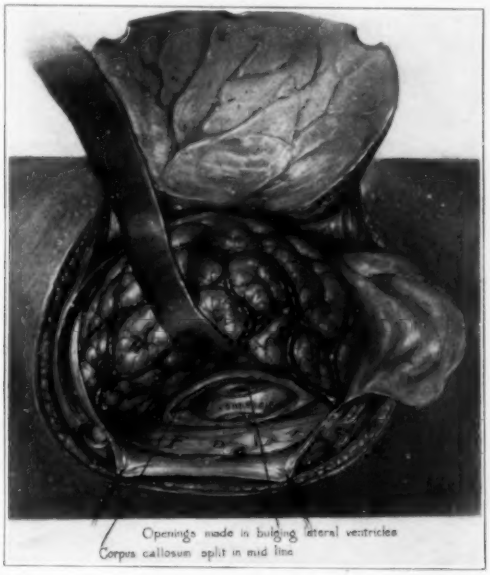

Fig. 14.—Operative approach to produce communication between the cavum septi pellucidi and the lateral ventricle on each side. The approach is along the midline of the head, anterior to the Rolandic vein. The hemisphere is separated from the falx, and the corpus callosum is split; the lateral ventricle can be seen bulging into the cyst of the cavum septi pellucidi.

Operation.—On May 28, 1929, a small bone flap was turned down on the right side of the head, slightly anterior to the rolandic area. The mesial side of the flap was directly over the longitudinal sinus. The dura was reflected toward the midline, and the right frontoparietal part of the brain was retracted from the falx. The corpus callosum was exposed and was found to be pushed upward; it was paler and thinner than normal, owing to the underlying pressure. An anteroposterior opening, about 2 cm. in length, was made in the anterior half of the corpus callosum (fig. 14). About an ounce of clear colorless fluid escaped as soon as the corpus callosum had been incised. The cavity from which the fluid was evacuated was everywhere smooth and glistening. Its color was essentially the same as that of the normal ventricular wall, but no blood vessels could be

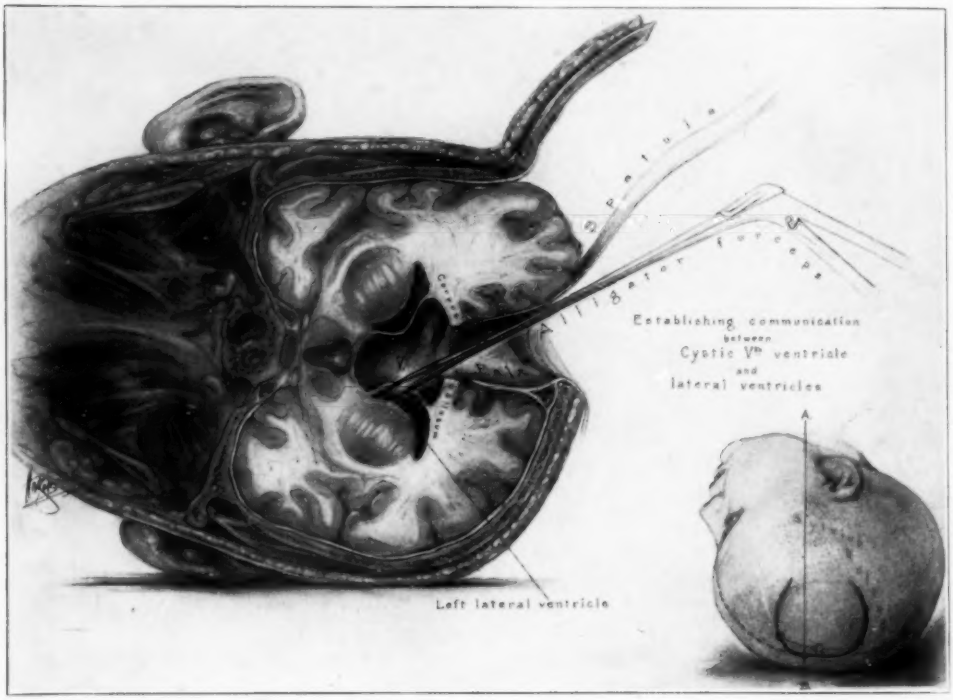

Fig. 15.—The method of producing an opening between the lateral ventricle and the cystic cavum septi pellucidi. Through this approach an opening can be made into each lateral ventricle.

seen. There was no visible choroid plexus. No openings of communication could be observed, and there was no sign of a papillomatous tumor. The cavity was oval. The following measurements were estimated: length (anteroposterior), 7 cm.; depth, 5 cm., and width, 5 cm. Symmetrical, smooth, longitudinally directed, rounded, bulging projections were present in each side. Each congenital projection was probably 0.75 cm. in depth and 1.5 cm. around the surface. These were the right and left lateral ventricles. With forceps an opening about 0.75 cm. long was made into each lateral ventricle (fig. 15). Cerebrospinal fluid then escaped from each lateral ventricle. The walls were opaque and about 3 or 3 mm. in

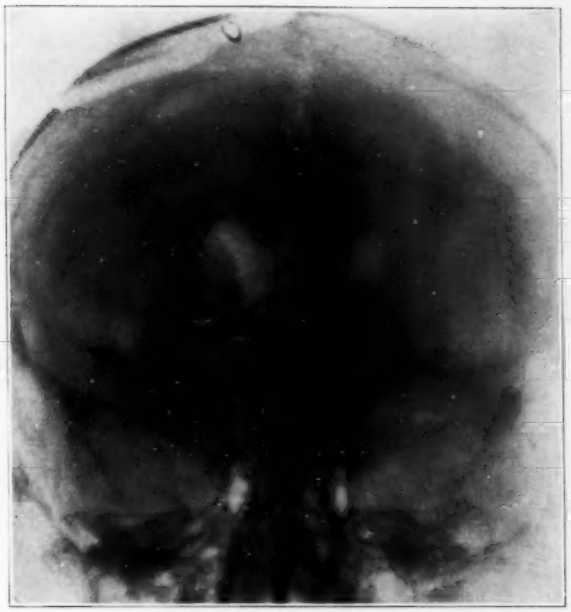

Fig. 16.—Anteroposterior ventriculogram taken after the operation. The air can be seen in the cavum septi pellucidi which now is in free communication with the lateral ventricles.

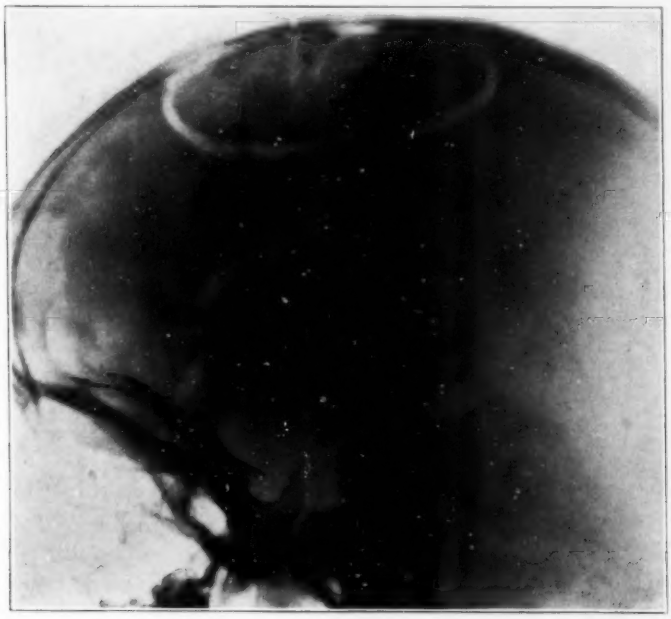

Fig. 17.—Lateral ventriculogram of the same patient as in figure 16. The location of the operative defect can be seen above the ventricle.

thickness—much thicker than the normal transparent wall of the septum pellucidum. The exposed surface of the brain showed a marked deformation of the cortical veins, and, doubtless, therefore, of the convolutions. There was no sylvian vein. The rolandic vein was formed just mesial to the sylvian fissure by confluence of five tributaries draining the frontal, temporal, parietal and occipital lobes (fig. 18). Soon after the rolandic trunk was formed, at a point about midway between the sylvian fissure and the longitudinal sinus, the vein curved sharply backward, then mesially again to enter the longitudinal sinus in approximately the normal position. In its course over the hemisphere the rolandic vein therefore formed roughly a semicircle with a radius of about 5 cm. instead of the

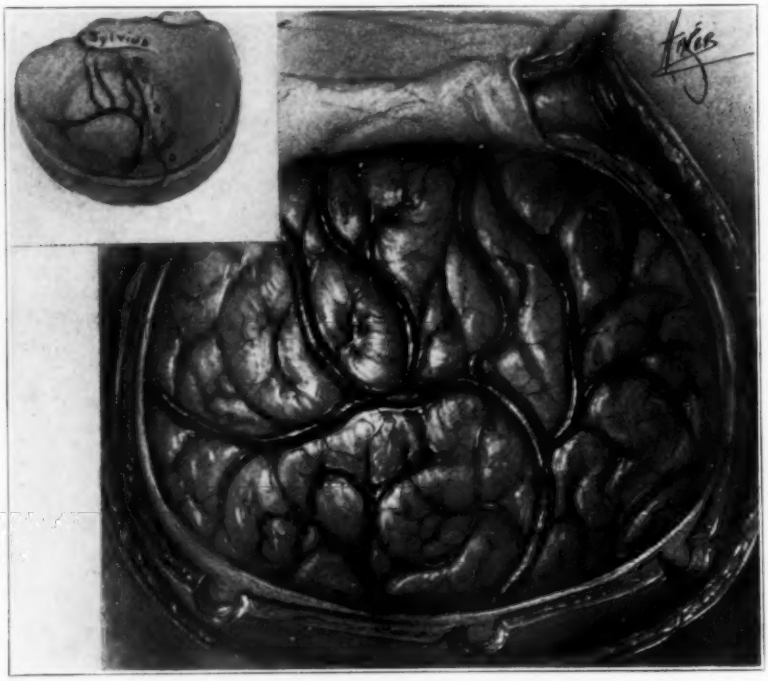

Fig. 18 (case 2).—When the hemisphere of this patient was exposed at operation marked deformation of the cortical veins was seen.

normal direct oblique course across the brain. The rolandic vein was much larger than normal, large enough, in fact, to suggest an arteriovenous aneurysm, but this possibility was excluded by the small tributary veins and by the absence of arterial blood in the vein.

There was no evident intracranial pressure. Room for the operative exposure along the corpus callosum was obtained by releasing the fluid by a ventricular tap (right ventricle). In view of my distressing experience in the preceding case the left anterior cerebral artery was carefully avoided.

The postoperative course was uneventful, the patient returning home two weeks later (fig. 19).

Subsequent Course.—The patient returned for observation eight months later. He had been perfectly well in every way. There had been no convulsions or attacks of paralysis. The Babinski reflexes were no longer positive. He appeared much brighter. His mother said that he was entirely well and much different than at any time preceding the operation. He was much more active and played with other children, whereas he had always been listless. The mental transformation seemed even greater to the mother. He was eager to learn, constantly repeating what he heard and talking of what he saw; previously he had been unconcerned. His memory was also greatly improved.

Critical Review of Case 2.—It is difficult to evaluate the clinical manifestations of this lesion except by postoperative changes, particularly as the patient had another congenital lesion, the vascular and convolutional deformations. The latter alone might cause convulsions,

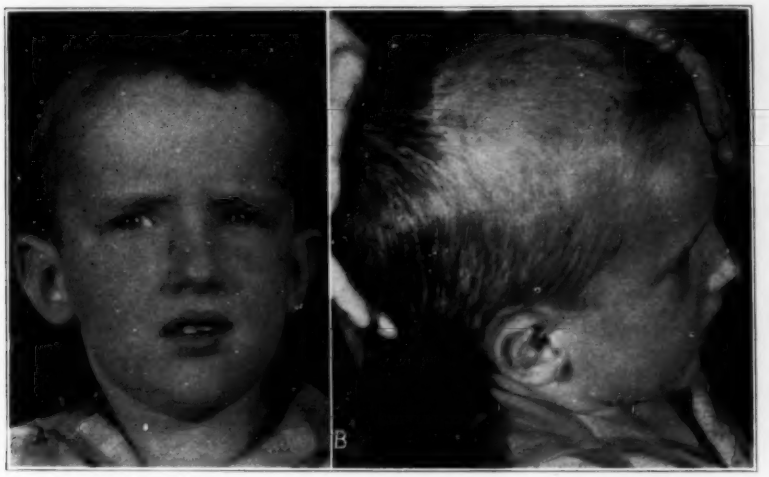

Fig. 19 (case 2).—*A*, patient at time of discharge from hospital; *B*, site of operation in same patient.

even unilateral convulsions, but not the convulsions of the variable character manifest in his case. He had some unilateral attacks, but others in which both legs and only one arm jerked. To produce these there must have been a mesially placed lesion in the brain. Moreover, they would not cause paralysis following the convulsions, nor a positive Babinski sign on both sides. Because of the recurring hemiplegia, the bilateral Babinski sign after the attacks, and the attacks affecting both legs but not the rest of the body, I had made a probable clinical diagnosis of an angioma near the midline of the brain, or possibly a cyst with an intracystic tumor. There had to be a gross tumor-like lesion to produce the signs of intracranial pressure, i. e., headache, vomiting, stupor and unconsciousness long after the convulsions were over. The

recovery of consciousness and the relief from hemiplegia suggested a lesion that varied in size over short periods of time, as for example a cyst. The exact type of lesion was not suspected until ventriculograms were made. The diagnosis was inescapable.

How far the mental and physical improvement was due to the cure of the lesion can only be conjectured. Mental impairment may indeed be due to malformation of the left cerebral hemisphere, such as that seen at operation in the right hemisphere. The patient was right-handed, however, and the actual malformation of the right hemisphere was of no concern in the mental make-up. Explanation of the mental backwardness on the basis of a cerebral deformation could therefore be possible only by assuming another malformation of the left cerebral hemisphere. The mental improvement that resulted following the permanent opening in the septum lucidum appears to be too prompt and too great to be unrelated to the dilated cavity, particularly since during the preceding years there had been no such spontaneous improvement, even for a brief interval. I am led to believe that much, if not all, of the mental deficiency may have been dependent on the large tumor. As the lesion lay on the aqueduct of Sylvius (and third ventricle), occlusion (of the ball-valve type) of the ventricular outlet from the third ventricle led to enlargement of both lateral ventricles and probably to enlargement of the head. The lesion was, of course, of congenital origin and the enlargement of the head was noticed soon after birth. This leads me to believe that the obstruction was only partial for several years, after an initial more complete blockage.

CLINICAL FEATURES

The only symptoms that stand out in these cases were headaches and mental aberration, and neither of these symptoms was characteristic of any condition. One could only think that there must be an organic lesion of some kind and that the trouble was sufficiently serious to demand the application of every possible diagnostic aid. That an organic lesion must exist was evident from the positive Babinski sign in each case and by the motor paralysis in case 2. The intermittency of the symptoms is also doubtless significant and can surely be explained by ball-valve action of the tumor and also because of its cystic character, which denotes variability of volume. That a congenital lesion should exist for forty years before giving trouble is surprising, but scarcely more so than the fact that once headaches developed they should disappear entirely after five years and not reappear after another five years. Moreover, experience with other large congenital space-occupying lesions of the brain, such as angiomas and arteriovenous aneurysms, is not dissimilar. They may exist for many—even forty or fifty—years

without causing any or many disturbances of function. As the mental disturbance in case 1 could not be due to syphilis, and as there was no reason to suspect arteriosclerosis as a cause, the diagnosis or exclusion of a new growth was demanded the more. In case 2 the mental retardation might well have been considered to be due to a congenitally malformed brain, as the patient had never been normal, but the period of coma could hardly be explained on any basis except intracranial pressure.

In neither of these cases was the location or the character of the lesion suspected until the ventriculograms were made. In case 2 the diagnosis was made before operation solely because of the experience derived from the ventriculograms and operation in the case 1. The only other lesion that could give a similar ventriculographic picture is a neoplasm in the region of the corpus callosum; in this instance the symptoms would be more progressive, less fluctuating and doubtless most fulminating.

Whether the incidence of these cavities in normal persons is as high as in the mentally deranged cannot be answered. There is no reason to believe that the psychopathic changes evident in most of the cases reported in the literature are at all dependent on the presence of these cavities. It seems probable that they denote a general trend toward cerebral malformations (there was evidence of this in one of my cases), and that the mental disturbance is the effect of these. As the cyst grows sufficiently large to cause signs and symptoms of local pressure, mental symptoms are then inevitable, because the part of the brain compressed is most concerned in mentality. If my single case of improved mentality after operation can be accepted as evidence, it would indicate that the cyst still remaining but collapsed, was without effect.

Ferrario's original case, which Verga confessed to be the best he had seen, presented no mental symptoms. Recently I noticed both of these cavities in several postmortem examinations of patients who appeared to be normal mentally.

SUMMARY AND CONCLUSIONS

1. Two cases of cysts of the cavum septi pellucidi and cavum vergae are reported. In each case the two cavities were continuous.

2. The cysts acted as tumors and caused compression of the motor tracts on both sides. Mental symptoms were decided in both cases. One patient had peculiar epileptic attacks of varying character but indicating bilateral involvement of the motor tracts. Suggestive evidence of intermittent intracranial pressure existed in both instances.

3. The diagnosis of cysts of this type cannot as yet be made by clinical signs and symptoms.

4. The diagnosis is easily made by ventriculography. The ventriculographic picture appears to be pathognomonic.

5. An operation is offered for cysts of this character. One patient was well and apparently normal eight months after operation. His physical and mental status was entirely changed by correction of the underlying cause.

ABSTRACT OF DISCUSSION

Dr. Ernest Sachs, St. Louis: Is there a definite cyst wall? About five years ago I took out an aspirated cyst which I thought was in the choroid plexus. It contained clear fluid which did not coagulate. Some of the cyst wall I sent to Dr. Bailey and he did not think that it was this kind of a cyst; at least he did not mention it.

Dr. Percival Bailey, Chicago: I did not think about this kind of a cyst, because I had never heard of it. I was interested to know what kind of fluid was in these cysts, and I am sorry that Dr. Dandy is not able to tell us, because, of course, the cavum septi pellucidi and the cavum vergae are both exterior to the brain, and one might expect them to be lined with connective tissue.

There is one other lesion that can give practically the same clinical picture that Dr. Dandy has described: the colloid cyst. From scrutiny of the encephalogram it might possibly be differentiated from this lesion because, theoretically, the colloid cyst should be above the corpus callosum instead of below. As I remember the literature, however, it is actually below, just under the anterior end of the corpus callosum, as were the cysts described.

Which anterior cerebral artery does Dr. Dandy think has to do with consciousness? In a recent case I amputated the right frontal lobe and tied the artery; the patient recovered consciousness rather promptly.

Dr. Charles R. Ball, St. Paul: How were the encephalograms made, by the ventricular or the lumbar method?

Dr. Walter Freeman, Washington, D. C.: I have seen a number of sections of the brain that have shown some dilatation of the septum pellucidi and occasionally one in which the cavum vergae was a little larger than ordinarily. I have never found what I would call a cyst wall in these cases. There were never any vessels on the interior and the fluid was always clear and resembled the cerebrospinal fluid although no chemical examination was made.

Dr. Dandy: In answer to Dr. Sachs' question, in the first case I could not tell the color of the ventricular fluid, because it was slightly blood-tinged. The injections of air were made by the ventricular method, which we use exclusively if a tumor is suspected.

In answer to Dr. Bailey's question, ligation of the anterior cerebral artery on the right side causes no noticeable disturbance. The loss of the arterior cerebral artery on the left side causes total and permanent loss of consciousness. We had seen this in three or four cases, before the cause was recognized. The first case in which our evidence to this effect was conclusive was one of bilateral frontal tumor. The left frontal lobe was first removed without any effect. Three weeks later the right frontal lobe was removed, and after that the patient never regained consciousness; at autopsy we found that the left cerebral artery and not the right, had been clipped, as we had suspected at the time. Many times we have ligated the right anterior cerebral artery without any appreciable after-effect.

THE CLINICAL SYNDROME OF THE CORPUS CALLOSUM *

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Clinical neurology has contributed little to the knowledge of the corpus callosum. Embryologic, anatomic and pathologic studies have elucidated many doubtful points in the knowledge of this portion of the brain, but knowledge of the clinical symptoms produced by lesions of the corpus callosum is extremely fragmentary and uncertain. Syndromes have been described which purport to be characteristic of lesions in this region. These, however, fail to elucidate features that are peculiar to involvement of the callosum as distinct from other portions of the brain.

Recent experience with a case in which we were able to diagnose a tumor springing from the corpus callosum stimulated us to review our cases of this type in order to determine whether a syndrome could be defined which seemed clearly to indicate involvement of the callosal region.

REPORT OF CASES

Our first case is of interest because we were able clinically to diagnose an involvement of the corpus callosum on the basis of neurologic symptoms.

CASE 1.—*Tumor of the left frontal lobe and corpus callosum. Drowsiness, mental signs, hemiparesis and apraxia. Exploratory craniotomy. Death.*

History.—A white woman, aged 58, was admitted to the Graduate Hospital in the service of Dr. T. H. Weisenburg in December, 1928. She had been well until May, 1928, when she began to have severe headaches which were more or less continuous and localized to the left temporal region. After a month, these became more or less intermittent. In June, she noticed that her right arm was weak, and she wrote her daughter that she was afraid she would lose the use of this extremity. In September, she became very drowsy and slept all the time, except when attending to household duties. She would get up, cook a meal and then go back to bed and sleep until the next one. In the last few weeks of the illness the family had noticed a change in personality, manifested by great irritability, loss of memory and a lack of interest in the family.

* Submitted for publication, June 2, 1930.

* From the Neurosurgical Service and its Laboratory of Neuropathology of the Hospital of the University of Pennsylvania and from the Services of Dr. T. H. Weisenburg and Dr. F. C. Grant, Graduate Hospital of the University of Pennsylvania.

Physical Status.—Physical examination revealed a fistula of the neck which lay 1 inch (2.5 cm.) to the right of the median line and about ½ inch (1.27 cm.) above the right clavicle. This discharged a yellowish material that proved to be due to cystic hygroma. The heart showed definitely weakened sounds, and the blood pressure was 134 systolic and 84 diastolic. There was slight edema of the ankles. Otherwise the physical examination gave negative results.

Neurologic Status.—Examination of the cranial nerves revealed a ptosis of both eyelids. On opening the eyes, the left palpebral fissure was smaller than the right. There were definite weakness of both external recti and difficulty in converging with the left eye. At times the patient complained of seeing double. She was weak in all extremities, but the right hand seemed slightly weaker than the left. She showed some dyssynergia with the right arm. She walked with a shuffling gait, but without a stagger. The deep tendon reflexes were all exaggerated, and there was a bilateral Babinski response. Further careful examinations were impossible because the patient was unable to cooperate properly.

The mentality was of interest. Most of the time the patient was stuporous. She lay in bed with the eyes closed and could hardly be aroused. At times she opened the eyes, but quickly returned to sleep as soon as she was left alone. It was extremely difficult to keep her attention even when the eyes were opened and she seemed to be cooperating. Sometimes she answered questions. Usually she paid no attention to them, but preferred to be left alone so that she might sleep.

At two examinations she showed a definite apraxia. When she was given a pencil and asked to demonstrate its use, she took it in her hand properly but could not employ it correctly. She was given a match and a match box, but she could neither tell what to do with them nor use them properly herself. She was given a finger-nail file, but could not tell how to use it. She held it in her hand like a pencil, and said that it was to write with. When given a comb she said the same, and when told to demonstrate its use she could not do so. Once when given a finger-nail file and told to show how to use it, she ran it through her hair. Further examinations of this sort could not be carried out because the patient's attention could not be held for very long at a time, and because she sank into a deeper and deeper stupor.

An examination of the eyes showed a choking of 3.5 diopters in the right eye, and 2.5 diopters in the left. There were hemorrhages in the retina.

A roentgen examination of the skull showed some evidence of increased intracranial pressure, with scattered areas of decalcification which were looked on as arteriosclerotic in origin.

The spinal fluid pressure was 34 mm. of mercury; the fluid contained: 7 cells, markedly increased globulin, 60 mg. of sugar and 650 mg. of chlorides. The Wassermann reaction of the spinal fluid was negative. Blood chemistry tests gave negative results, and a blood sugar curve indicated a decreased tolerance to sugar.

Course.—A diagnosis of tumor of the corpus callosum involving the left frontal lobe was made, and the patient was operated on by Dr. Francis C. Grant, who found the tumor springing from the corpus callosum.

Comment.—There was little question in this case that we were dealing with a tumor of the brain. The evidences of increased intracranial pressure, the elevated spinal fluid tension and the choked disks indicated this without much question. The question of localization was difficult, however. The stupor of the patient made examination difficult, and

the evaluation of the symptomatology and signs was always overshadowed by the mental obtundity of the patient. The outstanding facts were a history of weakness in the right arm noticed by the patient herself, stupor which was marked on entrance and became more and more pronounced, a relative objective weakness of the right arm, definite motor apraxia, a bilateral Babinski sign and weakness of the extra-ocular muscles as shown by a bilateral abducens weakness, bilateral internal rectus weakness and a paresis of upward convergence. The explanation of the bilateral Babinski response and the extra-ocular palsies was difficult when taken in conjunction with the other clinical manifestations. The presence of the pronounced mental symptoms, which will be discussed in detail later, the subjective and objective evidence of a right hemiparesis and the demonstration of a motor apraxia fitted into the syndrome of involvement of the corpus callosum as it had been described, and so a diagnosis of tumor of the corpus callosum was made. The severity of the signs could not be explained by this alone, so it was thought that the left frontal lobe was also involved. An operation disclosed a large cystic tumor in the left frontal lobe which had involved the callosal fibers in their anterior portion. This tumor extended into the genu of the callosum and involved it to a large extent.

The next case showed a similar clinical picture. The diagnosis was not made during life.

CASE 2.—*Tumor involving the genu and anterior third of the body of the corpus callosum. Mental signs and left hemiparesis. Operation. Death.*

History.—A white man, aged 37, entered the University Hospital in November, 1928, on the service of Dr. C. H. Frazier. He had been well until two months before entrance when headaches developed which were usually frontal, but which sometimes appeared in the occiput and radiated to the vertex. At the same time vision became blurred. He experienced vertigo on leaning forward, and at times felt as if he were going to fall to the left. He complained of projectile vomiting and a bad taste in his mouth, but otherwise of nothing else.

Neurologic Status.—An examination of the cranial nerves revealed a visual acuity of 6/22 in each eye, irregular pupils, sluggish convergence with each eye and a choked disk of 3 diopters in each eye. There was definite weakness of the left arm, the grip of the left hand being 25 as compared with 95 Kg. on the right. There was some slight dyssynergia of the left arm, with a tendency to stagger to the left and occasional swaying in the Romberg position. There were slow and uneven nystagmoid movements on looking laterally to either side, and some uneven rotatory movements on looking upward. The patellar reflexes were increased on both sides. Plantar stimulation caused flexion of the toes on both sides.

The mental symptoms were of interest. The patient was drowsy most of the time. He was dull, slow to grasp things and gave the impression of being very stupid. His mind wandered a great deal, and it was noted that he forgot to answer some questions that were put to him. His memory was poor. He was slow in making decisions.

The spinal fluid pressure was 220 mm. of water. The Bárány test pointed to a supratentorial lesion. Roentgen examination of the head gave negative results, and the pituitary fossa gave normal measurements.

Comment.—The evidences of dyssynergia were emphasized in this case, and the signs of a motor weakness were minimized. Consequently a suboccipital exploration was performed with entirely negative results. Following the operation, the patient progressed poorly and died. At

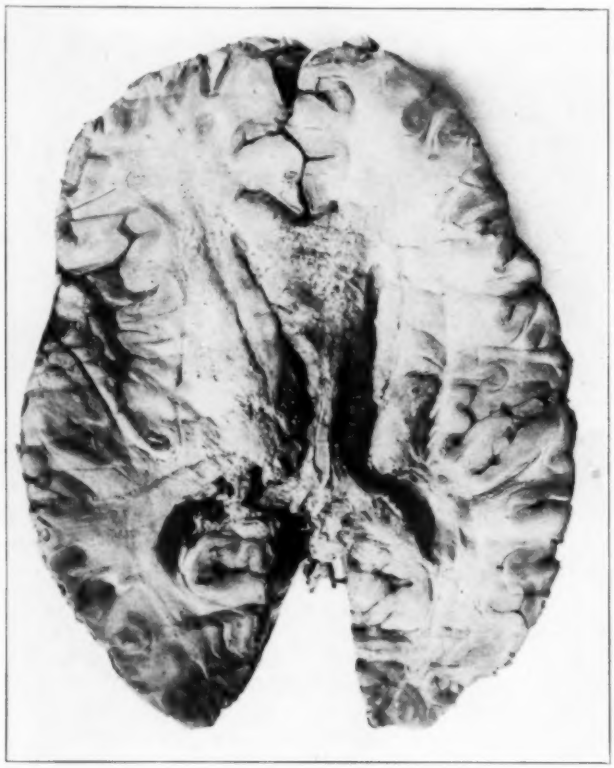

Fig. 1.—Tumor of the corpus callosum practically confined to this structure.

necropsy, a tumor was found involving the genu and anterior third of the corpus callosum, spreading into the right frontal lobe, and projecting into the tips of the anterior horns of the lateral ventricles. On retrospect, it was apparent that the mental symptoms and the weakness of the left upper limb should have made us suspicious of an involvement of the frontal lobe, and possibly also of callosal involvement. The mental symptoms especially seemed of import. There was not only a difficulty in concentration, attention, comprehension and memory, but also definite disorientation. No effort was made to determine the presence of an apraxia.

The subsequent cases are cited in order to elucidate the clinical evidences of tumor of the corpus callosum.

CASE 3.—*Tumor of the genu of the corpus callosum and the right frontal lobe. Mental signs, left hemiparesis. Operation. Death.*

History.—A white man, aged 29, entered the University Hospital in January, 1927, on the service of Dr. C. H. Frazier. He had been perfectly well until seven weeks before entrance when he was awakened at night by severe pain behind the right ear. Since then he had had constant headaches, which were growing more

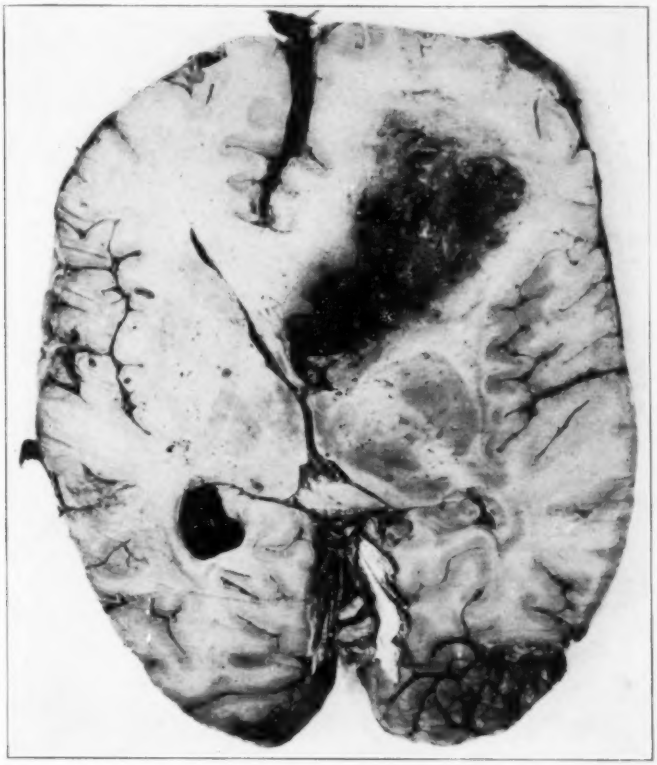

Fig. 2.—Tumor of the corpus callosum with extension into the frontal lobe.

intense. The headaches were described as bursting, tearing and crushing in character. About two weeks before entrance to the hospital, dizziness developed, followed by blowing and rushing sounds in the right ear.

Neurologic Status.—This showed a marked diplopia, weakness of both external recti, a slight deviation of the jaw to the left and a slightly smoother contour to the left as compared with the right side of the face. On slight effort the left side of the face was not as well innervated as the right, but on very strong effort both were equally well innervated. There was definite weakness of the left arm and leg. The grip of the left hand was 5 as compared with 35 Kg. on the right. The left leg was also weak. The right arm and leg were weak, but definitely stronger

than the corresponding members of the other side. The gait was unsteady, with a tendency to fall to the left. The left arm showed evidences of dyssynergia. There were slight twitchings of the left eyelid. The patellar and achilles reflexes were absent bilaterally.

There were definite mental symptoms. The patient was extremely drowsy and stupid. He understood everything that was said to him, but was extremely slow in executing commands. Often it was from thirty seconds to one minute between the command and the execution of the act by the patient. He himself had noted that his mind was affected so that he could not do things as promptly as he

Fig. 3.—Extensive tumor of the frontal lobes with involvement of the corpus callosum. Hemorrhage is present in both tumor areas.

wished. He had noticed a tendency to be tearful without any provocation. He was aware of a change in his personality as evidenced by a marked irritability. He had formerly been a cheerful person as compared with the irritable one examined. In the ward he complained of everything: the food, the bed and the confinement. He thought that his mind was affected and that everyone in the ward was conspiring against him. People were spying on him and discriminating against him. He noticed that his mind wandered a good deal, and that it was hard for him to concentrate.

There was 3 diopters of choking in each eye. Roentgen examination of the head gave negative results, and the measurements of the sella were normal.

Course.—The patient was operated on and died five days later.

Comment.—The outstanding features in this case were: the mental symptoms, consisting of inability to concentrate, poor attention, great difficulty in association and change in personality; the relative weakness of both the left arm and leg as compared with the right, and the extra-

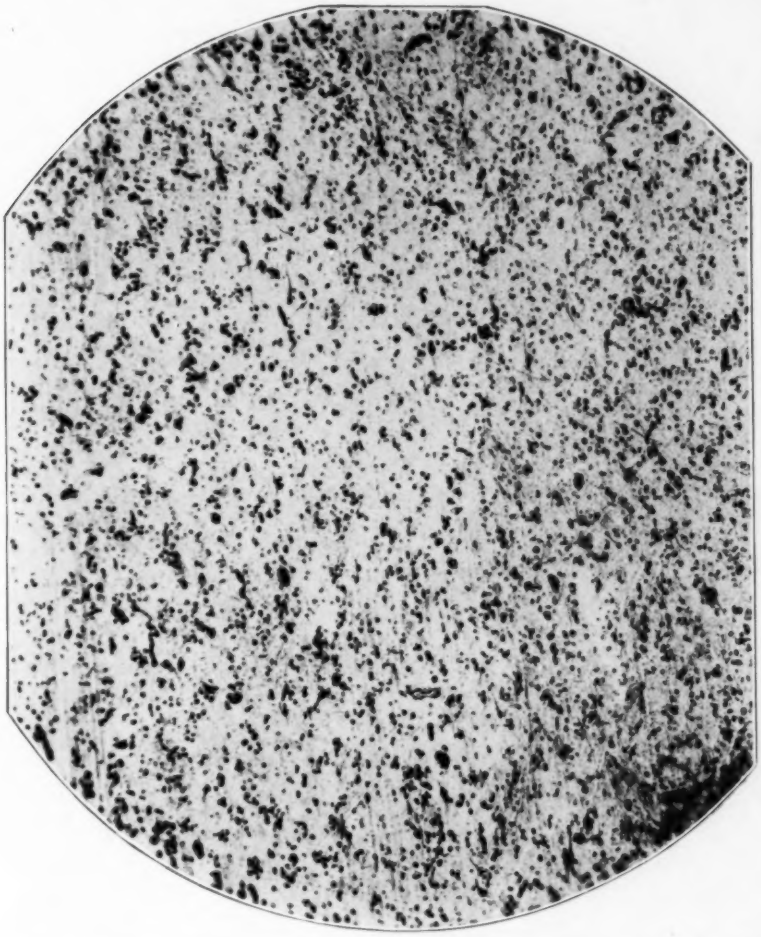

Fig. 4.—Tumor of the corpus callosum; numerous astrocytes are easily visible. Silver carbonate stain of Hortega.

ocular weaknesses consisting of a bilateral external rectus palsy. The significance of these for the syndrome of tumor of the corpus callosum will be discussed in detail later.

Case 4.—*Tumor of the corpus callosum. Mental symptoms, left hemiparesis. Necropsy.*

History.—A white man, aged 41, was admitted to the University Hospital in April, 1928, on the service of Dr. C. H. Frazier. For a year before entrance, the wife had noticed that he was dull and had difficulty in concentrating on his work, often forcefully beating on the table in order to keep his mind on his work. Sometimes he would fall asleep while eating, and would not talk unless questioned. He

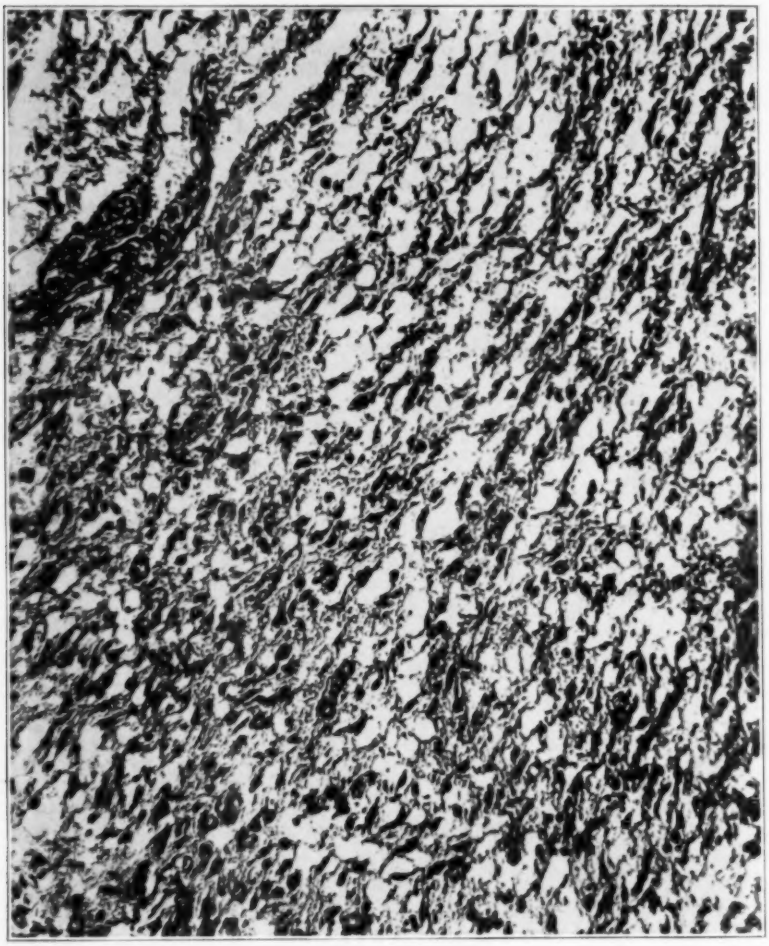

Fig. 5.—Tumor of the corpus callosum. The cells are arranged in a network of neuroglia fibrils. Many of them are astrocytes. Phosphotungstic acid-hematoxylin stain.

had usually been jovial and good-natured, but for the past year he had been dull. Three months before entrance, severe headache developed on the right side.

Neurologic Status.—The neurologic changes were few. The patient was weak in the left arm. He fell to the left, especially on walking a straight line. He had definitely dysmetria and past-pointing with the left arm. He also had sensory jacksonian attacks involving the left side of the face, arm and leg, which were

followed by drowsiness. He had weakness of both external recti. The patellar reflexes were increased on both sides.

Mentally, the patient was semistuporous. He could be aroused to answer questions briefly, but quickly lapsed "back into his sleepy and unconcerned condition." It was noted that his attention was poor.

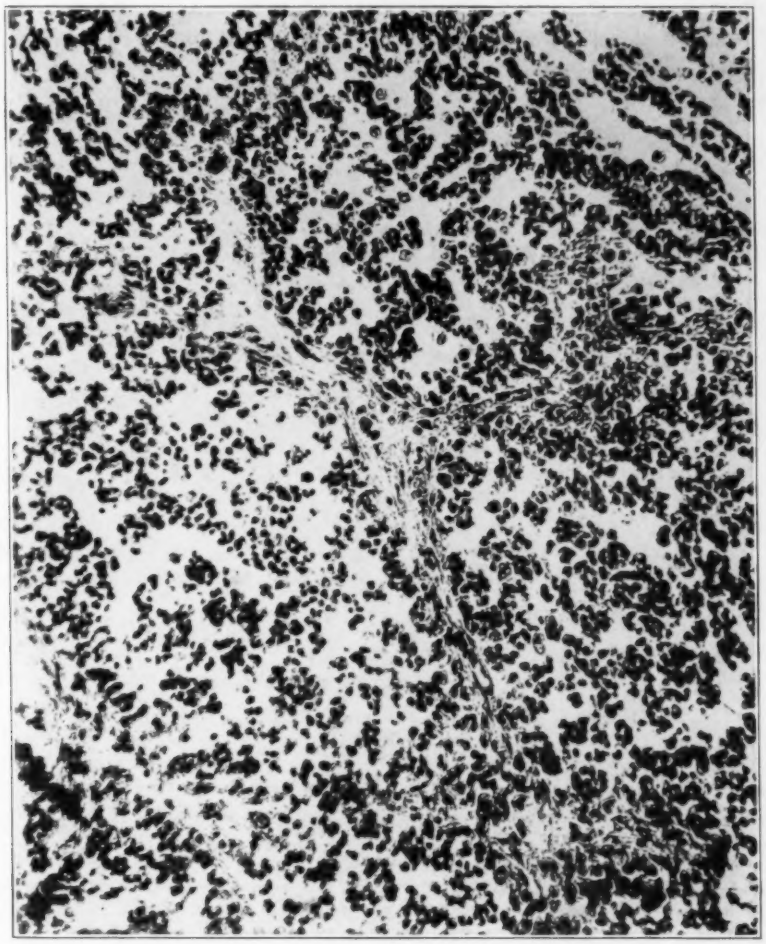

Fig. 6.—Tumor of the corpus callosum; the tumor is very cellular and tends to be arranged in lobules. Hematoxylin and eosin stain.

The spinal fluid pressure was 30 mm. of mercury. The eyes showed a swelling of 3 diopters.

Course.—A parietotemporal bone flap was reflected and a glioma exposed. The patient did not stand the operation well, and died three days later. Necropsy revealed a tumor involving the genu and anterior portion of the corpus callosum, extending also into the frontal lobe.

Comment.—The important features in this case were: a subjective complaint of lack of concentration, inability to think and drowsiness, with a slight change in personality; weakness and dyssynergia of the left arm; staggering to the left, and jacksonian attacks of a sensory nature involving the left side of the face and body. Both external recti were paretic. The objective mental manifestations consisted of lack of attention and a semistuporous state, accompanied by no loss of orientation.

CASE 5.—*Tumor of the corpus callosum. Mental signs. No motor weakness. Necropsy.*

History.—A white man, aged 19, entered the University Hospital in July, 1925, on the service of Dr. C. H. Frazier. He had been well until three months before entrance when generalized headaches developed. This was followed by impairment of vision. It was stated that he had become slow in thought, speech and movements during the three months before entrance.

Neurologic Status.—Neurologic examination showed a marked staggering on walking, a slight dyssynergia of both arms, more marked on the left, a slight ataxia and dysmetria of the right leg, slow thick speech and no motor weakness. Mentally, the patient was apathetic and expressionless. "He is slow in speech, movement, and cerebration. The mental development is that of a child of 10."

There was 6 diopters of choking in each eye, and a spinal fluid pressure of 16 mm. of mercury.

Comment.—The outstanding features of this case were the mental signs of slowness in cerebration and apathy. There were no localizing neurologic signs, save some vague evidences of cerebellar dyssynergia. Necropsy revealed a tumor involving the corpus callosum.

REVIEW OF THE LITERATURE

Tumor of the corpus callosum gives rise to a clinical syndrome which is at times clear and at others more or less obscure. The syndrome has been described by Levy-Valensi [1] and Raymond [2] and more recently by Mingazzini.[3] Since the cases of tumor in this region have been collected by various authors, it will not be necessary to review them in detail here. In 1902, Schuster [4] analyzed 32 cases; Lippmann [5] reported on the 50 cases collected to 1907, and Panegrossi [6] on 60 cases

1. Levy-Valensi, J.: Le corps calleux, Thèse de Paris, 1910.

2. Raymond, F.: Contribution à l'étude des tumeurs du cerveau, Arch. de neurol., 1893.

3. Mingazzini, G.: Der Balken, Berlin, Julius Springer, 1922.

4. Schuster, P.: Psychische Störungen bei Hirntumoren, Stuttgart, 1911.

5. Lippmann, H.: Zur Symptomatologie und Pathologie der Balkentumoren, Arch. f. Psychiat. **43**:128, 1907.

6. Panegrossi, G.: Contributo clinico ed anatomico-patologico allo studio, Policlinico **206**:238, 1908.

detailed prior to 1908. In 1910, Levy-Valensi [1] analyzed 93 cases from the literature, and Ayala,[7] in 1915, added 112 more cases which had not appeared in the studies of Levy-Valensi. Mingazzini [8] collected the cases up to 1922, and added 3 new cases of his own.

Levy-Valensi [1] formulated a syndrome of involvement of the corpus callosum consisting of negative signs, such as lack of involvement of the cranial nerves and of sensory disturbances, nonspecific characteristics in the form of disturbances such as drowsiness, coma, dementia, bilateral motor disturbances, sphincter disorders of the bladder and bowels, and dysarthria, and finally specific callosal disturbances in the form of mental signs and motor apraxia. The syndrome was stated a little more clearly and dogmatically by Milani,[8] who divided the manifestations of involvement of the corpus callosum by tumor into: (1) mental disorders; (2) motor apraxia, and (3) motor disturbances coming on late, slowly progressing, and involving one or both sides of the body.

The mental disturbances which both Levy-Valensi and Milani considered to be characteristic of involvement of the corpus callosum are of great interest. Their frequency has been emphasized by all investigators. Gianelli [9] found them in 100 per cent of tumors of the corpus callosum, and in the cases collected by Ayala they were present in every instance. Stern [10] has shown more recently, however, that tumor of the corpus callosum may exist without mental signs of any sort. He quoted cases reported by Schuster, Lippmann and Mills [11] and one of his own to demonstrate this point. Mingazzini has met this objection, however, by pointing out that mental disorders may occur very late in the syndrome instead of early, as is usually the case, and cited one of Stern's cases to show that mental signs did not appear until late in the disease when the tumor had reached a large size. The mental disorders of callosal involvement by tumor were well described by Raymond, who spoke of a definite "psychic syndrome" which occurred early in the disorder and was characterized by difficulty in association of ideas, loss of memory, especially for recent events, peculiar behavior, pronounced change in character and lability of emotions or temperament. In Raymond's conception, this syndrome was so characteristic that it needed no further accompaniment by other mental or neurologic signs to stamp

7. Ayala, G.: Contributo allo studio dei tumori del corpo calloso, Riv. di patol. nerv. **20**:449, 1915.

8. Milani, A.: Patologia del corpo calloso, Rome, 1914.

9. Gianelli, quoted by Mingazzini (footnote 3).

10. Stern, F.: Die psychische Störungen in Balkentumoren, Arch. f. Psychiat. **54**:663, 1914.

11. Mills, C. K.: Tumor of the Frontal Subcortex and Callosum, J. Nerv. & Ment. Dis. **36**:261, 1909.

it as due to involvement of the corpus callosum. The mental signs detailed appear early in the syndrome of tumor of the corpus callosum, but others appear later in the course of the disease due to the fact that the tumor is rarely confined to the callosum but spreads out into the surrounding brain substance.

The mental syndrome as analyzed by Ayala differs slightly from that of Raymond. In the early stages of the syndrome, it is characterized frequently by a depressed state—torpor, apathy and inhibition—often associated with memory disturbances, and without delusions or hallucinations. Later in the course of the disease, there occurs a very marked depression of faculties, interrupted at times by periods of excitement and passing over into stupor and coma. Stern has given a new conception, however, of the mental disturbances that occur in tumors of the corpus callosum. In his conception these are of two sorts: (1) Korsakoff's symptom-complex, and (2) a deficit in spontaneity of movement and thought. The former has been described by relatively few authors. The latter, the lack of spontaneity, is presumably due to the failure in the consecutiveness of associations caused by interruption of the fibers of the corpus callosum. Nevertheless, it is not peculiar to involvement of the corpus callosum because Stern pointed out that loss of initiative occurs particularly in involvement of the left frontal lobe.

Next in importance to the mental symptoms in the syndrome of tumor of the corpus callosum are the motor disturbances. These are characterized by the gradual onset of paresis of the limbs of one side, accompanied by indefinite motor symptoms of the opposite side, and without involvement of the cranial nerves. Bristowe[12] added to this description the fact that these signs are more marked in the lower extremities, are associated with contractures and eventually are accompanied by sensory disturbances and convulsions. Lippmann found motor disturbances in the limbs in 60 per cent of his cases, and Milani in 66 per cent. Levy-Valensi found it in sixty-eight of eighty-six cases. In forty-three cases the motor trouble was a flaccid hemiplegia, which came on suddenly. He did not find both sides of the body involved as frequently as did other investigators. It occurred in only thirteen of his ninety-six cases. Mingazzini and several authors before him expressed the belief that involvement of the cranial nerves does not occur in tumor of the callosum. Indeed, the lack of involvement of the cranial nerves and of the sensory tracts is one of the outstanding features of the syndrome of tumor of the corpus callosum. Ayala, however, has demonstrated that the cranial nerves are not infrequently involved, and that the abducens is particularly liable to injury.

12. Bristowe, C.: Cases of Tumor of the Corpus Callosum, Brain **7**:315, 1884.

The motor apraxia which is such an important feature of tumor of the corpus callosum will be discussed in detail later. Zingerle [13] described a "callosal ataxia" which he believed is characteristic of involvement of the corpus callosum. Other authors also believe that disturbances in equilibrium and gait are characteristic. This "callosal ataxia" consists in an inability of the patient to hold himself erect without support, and in an ataxic-spastic gait with a tendency to fall backward or to one side. Convulsions of a general and jacksonian type have been reported in many instances, but in many cases they have been absent from the clinical picture so that they do not constitute an integral part of the syndrome. The tendon reflexes are variable. Bristowe stated that they were not increased, but both Ayala and Levy-Valensi found that they were inconstant. In some instances they are increased; in others, decreased or normal. The Babinski response is often absent, but when present may be unilateral or bilateral. Sensory disturbances are usually absent, but Ayala reported a hemihypesthesia in four cases, hypostereognosis in one instance, pain on percussion of the skull in two cases, and pain on pressure over the supra-orbital branch of the trigeminal nerve in another case. Despite these observations, however, it is clear that in by far the great majority of instances tumor of the corpus callosum is not accompanied by sensory changes.

ANALYSIS OF MATERIAL

Our material consists of five cases involving the corpus callosum. In four of the cases the tumor had spread to the surrounding brain tissue, so that more than the corpus callosum was involved. In one case the tumor was confined to the corpus callosum. However, it is extremely rare to find a tumor confined to the corpus callosum itself, such instances having been recorded only in fifteen cases in the entire literature. These were cases reported by Berkley, Oliver, Beevor, Newton-Pitt, Pugliese, Alber, Fantoni, Mingazzini, Leichtenstein, Pick, Bergmann, Legrain and Marnier, Kopezynski, Legrain and Fasson, and Agostini (quoted by Mingazzini). In all five of our cases the tumor involved the anterior portion of the corpus callosum. In cases 1 and 2, the genu and the anterior portion of the body were implicated. In case 3, a similar distribution of the tumor was present, except that all of the genu and very little of the body were involved. In the two other cases, also, the corpus callosum was involved in its anterior portions. In all five cases the tumor involved the frontal lobe as well as the corpus callosum. In a few cases it had spread even farther than this and involved the temporal as well as the frontal areas.

13. Zingerle, Herman: Zur Symptomatik der Geschwülste des Balkens, Jahrb. f. Psychiat. **19**:367, 1900.

Mental Symptoms.—From the clinical standpoint the striking feature in all of our cases was the mental picture. All five patients presented mental symptoms. In three they were so pronounced as to be a striking part of the clinical syndrome, and in the other two they were less pronounced but none the less present. In case 1 the patient had shown at home a definite change in personality manifested by an irritability, which was in contrast to her usual good nature, and by a lack of interest in her family. She had become less conscientious about the care of her husband and children and less interested in their welfare. Objectively, she was semistuporous, but she could be aroused. When awakened it was difficult to keep her attention. She was totally unable to concentrate, was absolutely not interested in anything and showed a marked tendency to wandering of the mind. At times she was disoriented for place and time, but at others she was fully aware of her surroundings. It was almost impossible, most of the time, to center her attention on anything. She would answer a question briefly and then drop off to sleep, or pay no attention to the examiner. Examination under these circumstances was extremely exasperating, and had to be carried out piecemeal. The mental symptoms present in case 2 were characterized by difficulty in concentration, attention and comprehension, so that the patient gave the impression of being very stupid. His mind wandered a great deal and it was noted that he forgot to answer some questions. He was extremely slow in making decisions; his memory was poor and he was definitely disoriented for time. The patient in case 3 was extremely drowsy and was described as stupid. The mental symptoms were lack of attention and concentration, a definite change in personality, delusions of persecution and ideas of reference. It was often almost a minute between a request to do something and the execution of the act. He had noticed that his mind was unable to function as quickly as before the illness. The change in personality was manifested by a tendency to be very lachrimose on the least provocation, and by a marked irritability. He thought that everyone was spying on him and that people were conspiring and discriminating against him. The mental symptoms in case 4 were noted chiefly by the patient and his family, because when he entered the hospital he was semistuporous. He had noted mental dulness for a year before entering the hospital, and his inability to concentrate was sometimes so exasperating that he beat the table with his fists in order to keep his mind on his work. Sometimes he fell asleep at work. He had changed from a jolly to a dull person, and in the hospital it was noted that his attention was very poor. The patient in the last case showed "slowness of speech, movement, and cerebration."

Motor Disturbances.—These were not constant in all cases. In the first four cases there was weakness of one side of the body, while in

case 5 there was no demonstrable motor weakness. In case 1 the right arm and leg were definitely weak, a fact which the patient herself had noted during the course of the illness. However, the left side of the body was also weak, but was definitely stronger than the right. The clinical manifestations consisted, therefore, of weakness of both sides of the body, with definitely more weakness on one side. A similar condition prevailed in case 3, in which there was weakness in all the extremities, but more pronounced in one arm and leg than in the other. This bilaterality of the motor signs was early emphasized by Bristowe, but it is not constant. In cases 2 and 4 there was weakness of only one side, without corresponding weakness of the other side. In three of the four cases with motor weakness the arm was involved more than the leg. Only in case 3 did the weakness of the leg approach that of the arm of the corresponding side. In all cases with motor weakness, also, the face was either not at all affected or was so slightly weaker than normal as to appear of almost no import. This is striking in view of the weakness in the upper and lower limbs, and may be of some importance in indicating an involvement of the corpus callosum.

Drowsiness.—The patients in all five cases were extremely drowsy; four of them slept most of the time. The patient in case 1 slept all the time unless she was aroused, and then quickly dropped off to sleep again as soon as she was left unmolested. This is of interest in view of the tendency to associate sleepiness with lesions in the hypothalamus. In none of our cases, however, did the tumor extend to the hypothalamic region.

Cranial Nerves.—In three cases there was involvement of the ocular muscles. In case 1 there was a bilateral abducens weakness, and also a weakness of the oculomotor nerves indicated by an inability to look upward and to look inward to either side. The oculomotor weakness was not complete, except in the movement of the eyeballs upward, which the patient found impossible to carry out. In case 2 there was an inability to converge properly, and cases 3 and 4 showed weakness of both external recti muscles. In the other case there was no involvement of the ocular muscles. One case showed a slight weakness of the motor root of the trigeminal on one side with consequent deviation of the jaw. There were no other cranial nerve palsies in our series.

Cerebellar Signs.—Dyssynergia was present in some cases. In case 2 this was such a striking part of the picture that a diagnosis of cerebellar tumor was made and a suboccipital craniectomy performed. This patient had dyssynergia of the left arm and a definite tendency to stagger to the left side. In case 3 there was also slight dyssynergia of the left arm and a tendency to fall to the left. Case 4 showed some dyssynergia in one arm, while in case 5 there was marked staggering

on walking and slight dyssynergia of both arms, which was more marked on the left side. Except for case 2, however, the cerebellar signs were never very striking; in most instances they were so overshadowed by the mental and motor disturbances as to appear of relatively little value in the total clinical picture.

Evidences of Increased Pressure.—In three of the five cases, the onset of the trouble was with evidences of increased intracranial pressure, such as headache and vomiting. The spinal fluid pressure in our cases, measured in millimeters of mercury, was 34, 12, 30 and 16. In one case it was not recorded. The choked disks in case 1 measured 3.5 diopters in the right eye and 2.5 in the left; in cases 2, 3 and 4, the choking was 3 diopters in each eye, and in case 5 there was 6 diopters of choking in each eye. The choking, therefore, varied between 2.5 and 6 diopters. Usually it was about 2.5 or 3 diopters.

SYNDROME OF THE CORPUS CALLOSUM

The syndrome of the corpus callosum as seen in our five cases is clear, without being absolutely characteristic. The clinical picture consists of mental disturbances, motor paralysis, drowsiness in a great many cases and sometimes motor apraxia. Cerebellar dyssynergia is sometimes present, and there is often paralysis of the cranial nerves, especially of those innervating the ocular muscles. Evidences of increased pressure in the skull are frequent, are usually not very great and are attended with only a moderate degree of choked disk in most instances.

The onset of the clinical picture varies. Sometimes the first manifestations of trouble consist in mental disturbances, and sometimes in evidences of increasing pressure within the skull. The onset may be very sudden with severe headache, as in two of our cases, or may be slowly progressive. Whatever the mode of onset, however, the mental signs constitute an important part of the clinical picture. In all five of our cases the mental disturbances overshadowed all other signs that were present. They constitute the chief feature of the clinical picture. The most striking thing in the mental picture is the inability of patients with a callosal tumor to concentrate and to focus and maintain attention for even a short time. Their minds wander; they often forget to answer questions; they cannot grasp too complicated thoughts, and they are often so slow in cerebration that it takes from thirty to sixty seconds for them to answer a question. Often they give the impression of being very stupid because of this inability to think and associate quickly. They cannot remember things clearly, and fail in memory for recent events particularly. Sometimes disorientation is present, and, as in one of our cases, frank delusions of persecution and ideas of

reference may complicate the picture. The inability to concentrate may be equally apparent to both patient and friends; for example, one of our patients was so exasperated by the inability to keep his mind on his work that he beat the table in despair. Engrafted on all this may be a change in personality. This occurs fairly frequently, but is not as a rule very deep-seated. It consists usually of irritability in a previously happy person, or the development of an apathy or indifference in a person who was formerly keen and conscientious. In case 1 this had progressed to the point where a faithful mother became negligent of her house and children. Behind all these mental signs there is often a background of drowsiness which may be so extreme that the patient cannot remain awake for an appreciable length of time. A state of almost constant drowsiness is present from which the patient can be aroused, but into which he quickly lapses as soon as he is left to his own devices. The mental symptoms which we have observed to be most characteristic are the inability to concentrate and the apathetic indifference of patients. This has been present in all our cases, and striking in most of them. Failure to keep the attention fixed on whatever subject is at hand is frequent and is noted by the patient, by his friends and by the examiner.

In view of the fact that the fibers of the corpus callosum connect corresponding areas in the two hemispheres and also heterotopic areas (Ramón y Cajal,[14] Van Valkenberg [15]), thus creating rich association paths between many areas of the brain, they are probably of great importance in the ability to form associations and to bring into play various sections of the brain. A tumor interrupting these complex and rich connecting fibers interferes with the ability to use many portions of the brain. It is no surprise, therefore, that inability to concentrate and to keep attention focused on a subject is one of the striking and outstanding symptoms in tumor of the corpus callosum.

The mental symptoms are more pronounced in some cases than in others, and an effort has been made to explain this by postulating that such symptoms are more pronounced or are usually present when the tumor involves the anterior portion of the corpus callosum, and are either less pronounced or absent in tumors involving the posterior portion. This cannot be demonstrated satisfactorily in every instance. Nor can cases of tumor of the corpus callosum without mental symptoms be explained away easily. Mingazzini concluded that there is no relation between the occurrence of mental symptoms in tumor of the corpus

14. Ramón y Cajal, S.: Textura del sisterna nervoso, Madrid, N. Moya, vol. 2, p. 845.

15. Van Valkenberg, C.: Experimental and Pathological Research on the Corpus Callosum, Brain **36**:119, 1914.

callosum and the situation of the tumor in the callosal system of fibers. While many cases have been reported in which mental symptoms occurred when the anterior fibers were involved, there have also been cases in which mental symptoms were present with implication of the splenium and posterior part of the body.

Experimental evidence indicates that mental symptoms occur in animals in which the corpus callosum has been sectioned. Lafora and Prados [16] mentioned mental signs among the features observed in apes and cats whose corpus callosum had been sectioned on either side of the falx cerebri. More recently, Seletzky and Gilula [17] found definite mental signs after cutting the callosal fibers in cats and dogs. These symptoms were found to occur mainly on section of the genu and the anterior portion, and consisted of apathy and a state of inhibition characterized by inactivity and lack of interest toward everything that occurred around the animals. They were entirely inactive and uninterested. Periods of excitement sometimes occurred in the acallosal animals, and were accompanied by evidences of great anxiety, such as is shown by dilated pupils and erection of the hair. It is evident, therefore, that mental symptoms similar to those found in man are found also in animals with the corpus callosum severed, and that these symptoms are apathy and indifference, in both man and animals, plus a lack of concentration in man. To these may be added changes in personality and evidences of delusions and hallucinations in some cases.

Equally important, but sometimes not as striking as the mental signs, are the motor signs. These were present in four of our five cases. In two cases the weakness involved all four limbs, but was more pronounced on one side of the body, and in the other two cases the paresis was present on only one side of the body. The weakness is usually not very great, but is sufficiently pronounced to be evident on objective examination. Curiously enough, the face often escapes in involvement of the limbs in tumor of the corpus callosum, a sign which may have some diagnostic importance. In all our cases with hemiparesis or with weakness of all four limbs the face either was not affected or the implication was so slight as to appear of no importance in the clinical appraisal of the case. In the apes and cats operated on by Lafora and Prados, there occurred a hemiparesis and apraxia on the opposite side. It is interesting to note that in their experiments they found that lesions in the genu caused apractic symptoms in the forepaw

16. Lafora, S., and Prados: Experimentalversuche über die Funktion des Gehirnbalkens, Ztschr. f. d. ges. Neurol. u. Psychiat. **84**:617, 1923.

17. Seletzky, W., and Gilula, J.: Zur Frage des Balkens bei Tieren, Arch. f. Psychiat. **86**:57, 1928.

only; transection of the posterior half caused paresis and apraxia in the hind paw only, and that complete section of the corpus callosum caused a marked hemiparesis and apraxia. A point of great importance in the experimental work of Lafora and Prados and Seletzky and Gilula is that the symptoms caused by transection of the corpus callosum in cats, dogs and apes disappear after a relatively short time and leave no evidence of their former presence.

Finally, apraxia when present is an extremely important indication of involvement of the corpus callosum. Unfortunately, it is not often present in tumors involving the callosum. Mingazzini found only ten cases in the literature in which tumor of the corpus callosum was accompanied by apraxia. In other lesions of this region, as for example in softening, apraxia occurs more commonly. In the ten cases gathered together by Mingazzini, the apraxia was on the left side in most instances. In a few instances it was bilateral. Mingazzini believed that when apraxia occurs in the left hand in a person with tumor of the brain, the indication is undoubtedly that the corpus callosum is involved, provided of course a mental cause for the occurrence of the apraxia can be ruled out. In our case 1 it was possible to make a definite diagnosis of tumor of the corpus callosum because of the presence of apraxia. In the cases reported, the apraxia has been found in tumors involving the genu and the anterior half of the callosum. Our case supports this observation. The tumor diagnosed by us implicated the anterior portion of the corpus callosum. In our opinion the presence of apraxia points almost certainly to an involvement of the corpus callosum, especially if it is accompanied by hemiparesis and mental signs of the nature herein mentioned.

SUMMARY AND CONCLUSIONS

Our experience with five cases of tumor of the corpus callosum indicates that there is a clinical syndrome indicating involvement of this structure. This syndrome is most characteristic when the presenting symptoms include mental disturbances, hemiparesis and motor apraxia. A syndrome with the mental symptoms described and with hemiparesis and apraxia is almost characteristic of involvement of the corpus callosum. Less characteristic, but none the less indicative of callosal implication by tumor, is a syndrome consisting of pronounced mental symptoms with drowsiness and hemiparesis. In addition, there may be evidences of increased pressure and dyssynergia. It is hazardous to state that such a syndrome as that last mentioned is characteristic of disease of the corpus callosum, but in our experience the mental symptoms that have been found in cases of tumor of the corpus callosum

are so strikingly similar in all cases as to justify our stating that in cases with evidences of increased pressure, with mental signs of apathy, indifference, lack of concentration, change in personality and occasionally psychotic manifestations, occurring in the presence of a hemiparesis or even without, as in our case 5, tumor of the corpus callosum should be strongly suspected. The probabilities are that in such cases a tumor will be found in this location.

1. Five cases of tumor of the corpus callosum are reported. One of these was diagnosed during life.

2. A clinical syndrome of the corpus callosum is described.

SPASMODIC LATERAL CONJUGATE DEVIATION OF THE EYES

A CLINICOPATHOLOGIC STUDY *

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The subject of oculogyric spasm, better known as spasm of lateral conjugate deviation of the eyes, has received considerable attention. Descriptions of the anatomic, physiologic and pathologic aspects of the phenomenon given by various observers differ. We believe that the interpretation of the symptoms in a case will benefit from a brief review of the subject from this point of view.

ANATOMY

Present knowledge concerning the anatomy of oculogyric spasm and paralysis owes much to the works of Dejerine [1] and of Tilney and Riley.[2] The cephalogyric and oculogyric centers can be considered in two main divisions:

A. Cortical Centers.—These consist of: 1. The anterior or motor center, situated in the posterior portion of the foot of the second frontal convolution, from the cells of which cortical oculogyric and cephalogyric fibers belonging to the corticosegmental pathway take origin. The anterior center is associated with the peripheral centers by these fibers, which pass through the knee of the internal capsule, the internal portion of the pes pedunculi, descend with the aberrant fibers of the peduncular pathway adjacent to the mesial fillet and supply fibers (aberrant pyramidal) to the nuclei of the third nerve and to the opposite nuclei of the sixth nerve.

2. The posterior or visual center, situated in the vicinity of the calcarine fissure, from the cells of which fibers originate that form the

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1. Dejerine: Sémiologie des affections du système nerveus, Paris, Masson & Cie., 1926; Anatomie des centres nerveus, Rueff & Cie., 1895, vol. 2.

2. Tilney, F., and Riley, H. A.: The Form and Functions of the Central Nervous System, New York, Paul B. Hoeber, 1921.

visual corticofugal pathways going to the superior colliculus. The posterior visual cortex is associated with the peripheral centers by the corticofugal fibers which, intimately associated with the optic radiation, converge toward the posterior limb of the internal capsule, and, skirting the postero-inferior aspect of the optic thalamus, reach the superior colliculus (fig. 1). From there the corticomesencephalic oculomotor fibers (the best known of which are the oculogyric fibers) reach the vicinity or the level of the sixth nerve nucleus (the center for coordination of binocular movements), giving off fibers to the third, sixth and eleventh nerve nuclei and to the anterior horn cells of the spinal cord (fig. 1).

The cortical oculomotor centers of one hemisphere are united by association fibers that belong to the superior and inferior longitudinal fasciculus and the occipitofrontal fasciculus. The cortical centers of both hemispheres are united by commissural fibers that pass through the corpus callosum,

B. Intermediary or Supranuclear Centers.—Although there are definite clinical and anatomic facts pointing to the existence of the intermediary centers, their anatomy is not well understood.

1. The superior colliculus (anterior quadrigeminal body) is the principal center for the direction of movements of the eye, head and body in connection with the visual sense.

The visual oculogyric or tectospinal pathway, as already mentioned, takes origin from the cells of the superior colliculus, crosses the median line at the level of the dorsal tegmental decussation of Meynert † in the prelongitudinal fasciculus, and gives off terminal and collateral fibers to the homolateral nuclei of the third nerves, the opposite nuclei of the sixth nerves, the cephalogyric nuclei of the eleventh nerves and the anterior horn cells of the spinal cord.

The tectospinal pathway is stimulated in two ways: (1) by the peripheral visual pathways (optic nerve, chiasm and optic tract), the fibers of which go to the superior colliculus, lateral geniculate body and pulvinar, and (2) by the central corticofugal visual pathways which bring stimuli from the central visual cortex of the calcarine fissure to the superior colliculus.

From the cells of the superior colliculus, as already mentioned, impulses pass to the pons, medulla oblongata and spinal cord. The impulses from this center (superior colliculus) form the reflex command of looking in this or in that direction in order to hold the object in view.

† "This commissure establishes a complete crossing of the tectospinal and tecto-bulbar tracts. It makes possible a communication between the superior colliculus of the one side and the ventral horn cells and motor nuclei of the cranial nerves upon the opposite side." (Tilney [footnote 2], p. 538.)

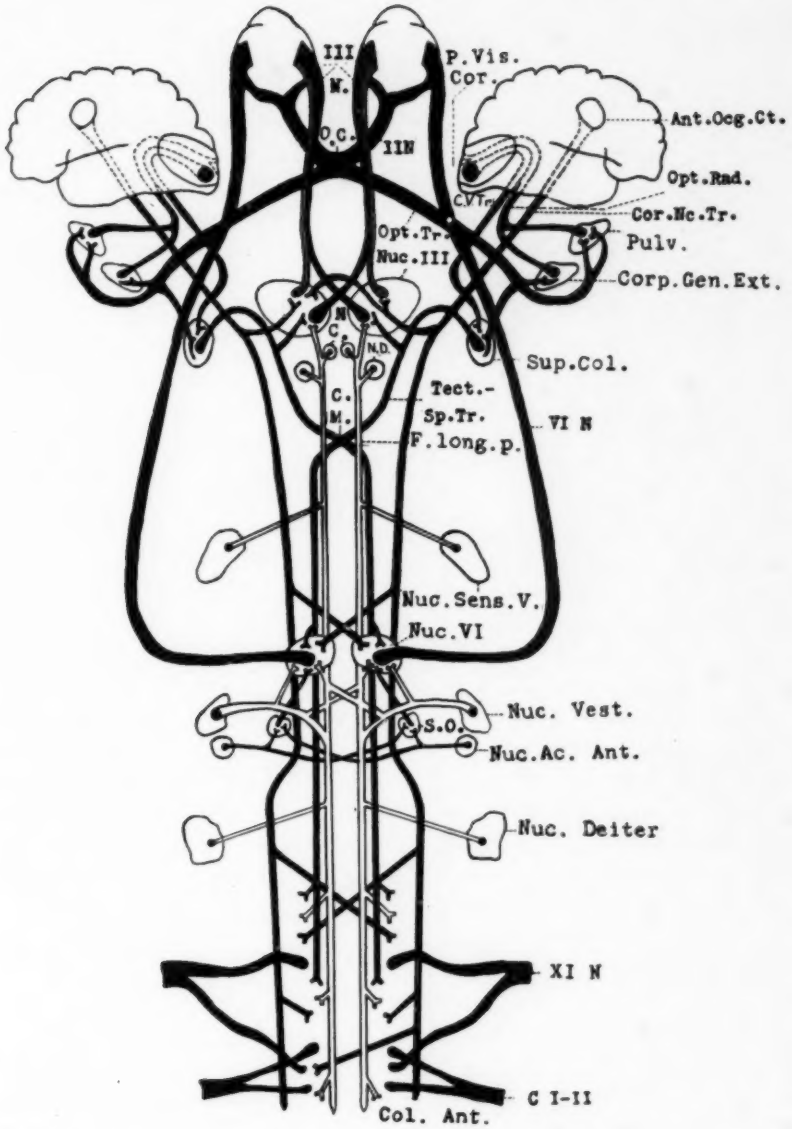

Fig. 1.—Diagram illustrating the oculogyric and cephalogyric mechanisms (modified after Dejerine). *II N.* indicates the optic nerve; *III N.*, oculomotor nerve; *VI N.*, abducens nerve; *XI N.*, spinal-accessory nerve; *Ant. Ocg. Ct.*, anterior oculogyric center; *C I-II*, first and second cervical nerves; *C. M.*, commissura Meynert; *Col. Ant.*, columna anterior; *Cor. Nc. Tr.*, corticonuclear tract; *Corp. Gen. Ext.*, corpus geniculatum externum; *C. V. Tr.*, corticofugal visual tract; *F. Long. P.*, fasciculus longitudinalis posterior; *Nuc. III*, third nerve nucleus; *Nuc. VI*, abducens nucleus; *Nuc. Ac. Ant.*, nucleus acusticus anterior; *N. C.*, nucleus interstitialis Cajal; *N. D.*, nucleus Darkschewitsch; *Nuc. Deiter*, nucleus of Deiter; *Nuc. Sens. V.*, nucleus sensorius trigeminus; *Nuc. Vest.*, nucleus vestibularis; *O. C.*, optic chiasm; *Opt. Rad.*, optic radiation; *Opt. Tr.*, optic tract; *P. Vis. Cor.*, posterior visual cortex; *Pulv.*, pulvinar; *S. O.*, superior olive; *Sup. Col.*, superior colliculus; *Tect. Sp. Tr.*, tectospinal tract.

2. The labyrinthine oculogyric connections consist of (*a*) the static or vestibular oculogyric pathway and (*b*) the acoustic or cochlear oculogyric pathway.

(*a*) The vestibular oculogyric pathway has its origin in cells of the superior and lateral vestibular nuclei. From these nuclei the fibers pass horizontally to the posterior longitudinal fasciculus of the same or opposite side and there divide in ascending and descending branches. The ascending branches end in the nuclei of the third, fourth and sixth nerves; the descending branches end in the nucleus of the spinal accessory nerve and the columna anterior of the cervical portion of the spinal cord. Through these pathways the reflex response of the muscles of the eye to afferent impulses arising in the vestibular and semicircular canals of the ear is made possible. Through Deiters' and Bechterew's nuclei the electrical, thermal and mechanical stimuli of vestibular origin are brought into direct relation with the third and sixth nerve nuclei, thus producing the reflex movements of the eyes known as nystagmus.

(*b*) The cochlear or auditory oculogyric pathway consists of fibers that originate in the cells of the anterior acoustic nucleus, the trapezoid body, the fibers going to the superior olive of the same side and the opposite one and the fibers that go to the lateral lemniscus. Auditory stimuli from the internal ear are thus transmitted from the pons to the midbrain by the lateral lemniscus, from which collaterals terminate in the inferior colliculus. From the inferior colliculus the impulses reach the third nerve nuclei, and thence, through the fibers going to the muscles of the eyeball, direct the eyes to sounds above or below the level of the eyes. By the reflex connection of the superior olive and the nuclei of the sixth nerves the eyes move to the right or left in response to sounds.

It will be seen that the labyrinthine oculogyric pathway can be stimulated not only by the vestibular and cochlear peripheral sensations, but also by the cerebellum through the internal semicircular and cerebellovestibular pathways which end in Deiters' and Bechterew's nuclei.

While the superior colliculus forms the reflex center for ocular movements, the labyrinthine oculogyric connections form the coordinating center, which subserves a double purpose: the coordination of binocular movements and general coordination, such as equilibrium and general orientation.

3. The trigeminal oculogyric pathway originates in the nuclei of the fifth nerve, dividing into ascending and descending branches which form connections with the nuclei of the third and sixth nerves, the cephalogyric nuclei of the eleventh nerves and the columna anterior of the cervical portion of the spinal cord. Through this pathway, lateral oculogyric movements of the eyeballs can be induced by pinching the skin of the head or by stimulation of the conjunctiva of the eyeball.

4. The interocular associated reflexes are regulated by the posterior longitudinal fasciculus. This pathway, going from the anterior horn cells of the spinal cord to the thalamic and subthalamic region, is made up of: (1) ascending fibers coming from (*a*) superior and lateral vestibular nuclei; (*b*) Deiters' nucleus, by means of which a connection is established between the third, fourth and sixth oculomotor nerves, bringing them under direct influence of the cerebellum and equilibratory mechanism; (*c*) fibers from the sensory portion of the fifth nucleus, correlating the movements of the eye with sensory impressions received on the conjunctiva and head; (*d*) fibers from anterior horn cells of the spinal cord; (*e*) fibers from cells in the reticular formation of the bulb, pons and midbrain; (*f*) fibers from the fourth and sixth cranial nerve nuclei; (*g*) fibers from the nucleus of Darkschevitch. (2) The descending fibers of the fasciculus longitudinalis posterior are derived from: (*a*) the nucleus interstitialis of Cajal (a collection of cells situated mesial to the cephalic extremity of the red nucleus); (*b*) the superior and lateral vestibular nuclei; and (*c*) from the third and fourth cranial nerve nuclei.

As is readily seen, the fasciculus longitudinalis posterior is the great reflex pathway which brings into coordinative relation the three nuclei in the oculomotor mechanism, i. e., the third, fourth and sixth cranial nerve nuclei. Impulses originating in the sixth nerve nucleus pass by way of the fasciculus longitudinalis posterior to the fourth and third nerve nuclei, stimulating responses appropriate to produce the necessary associated interocular reflexes. The fasciculus longitudinalis posterior also brings to the three coordinating oculomotor nuclei the involuntary stimuli coming from the visual, auditory, vestibular, cerebellar and general sensory apparatus. Furthermore, the sixth nerve nucleus receives a bundle of fibers from the superior olive, which probably serves to join functionally the motor apparatus for the direction of movement of the eyeball to the auditory and vestibular apparatus.

C. The Primary or Peripheral Centers.—These consist of (1) the primary nuclear centers and (2) the peripheral nerves.

The centers are formed by: (*a*) the oculomotor nucleus and its nerve. From the cells of the anterior and median nucleus, fibers go to the intrinsic muscles of the eye, the majority of which are homolateral or direct, while those supplying the internal and inferior recti as well as the inferior oblique are crossed. The nuclei of the third nerve have association fibers that run from each nucleus to that of the opposite side. (*b*) All fibers of the nucleus of the fourth nerve cross, and as this nerve is concerned with upward movement of the eyes, we shall not describe it in detail. The nuclei of the fourth nerve are not connected with each other. (*c*) All fibers of the nucleus of the sixth nerve are

direct and supply the external rectus muscle. These nuclei are not connected with each other.

PHYSIOLOGY

Disturbances in lateral conjugate deviation of the eyes are not to be attributed to lesions of the muscles of the eyes, the nerves or the primary nuclei, but to lesions of the coordinating mechanism which is charged with the division of labor of the different peripheral motor elements. The eyeballs can move together either to the right (dextro-ocular function) or toward the left (levo-ocular function).

The lateral conjugate deviations are more complicated than the upward or downward movements. In the latter the eyes are displaced in one common plane, the horizontal, while in the former each eye is displaced in both the vertical and the anteroposterior plane, which is different from that of the opposite eye. The lateral displacement does not take place in relation to the median plane of the body, but in relation to the anteroposterior axis of each eye. This can best be illustrated by analyzing the movements of the eye, for example, when looking to the right. This is accomplished by the contraction of the external rectus muscle of the right eye and the internal rectus of the left eye through the action of the primary motor nuclei of these two muscles. The two primary motor nuclei are coordinated for the turning of the eyes to the right by the coordinating mesencephalic center placed to the right of the median line and called the dextro-oculogyric center. This center directs the right external rectus of the same side and the right internal rectus of the opposite side. This coordinating center is in connection with the left cortex by tracts and pathways which have already been described. Therefore, a command brought from the left cortex will carry the two eyeballs to the right. This entire corticomesencephalic mechanism of binocular turning of the eyes to the right was called by Grasset [3] the dextro-oculogyric apparatus, that of turning the eyes to the left, the levo-oculogyric apparatus. Summarizing, it can be stated briefly: The right cortex turns the eyes to the left, and the left cortex turns the eyes to the right. As Grasset [3] said, "Each hemisphere sees and looks to the opposite side." This can be emphasized further by stating that visual, acoustic, equilibratory and sensory impressions bring reflexes to one of the two coordinating centers. The association pathways between these ocular functions is the fasciculus longitudinalis posterior. While these statements are true physiologically, certain variations occur in the operation of this mechanism depending on whether the lesion is paralytic or irritative. This will be brought out in the discussion of the case.

3. Grasset, J.: De la déviation conjugée de la tête et des yeux, V. A. Delahaye, Paris, 1879, pp. 1-24.

Although the eyes can be moved laterally without any turning of the head, these two movements are nevertheless closely associated. The cephalogyric centers and pathways are close to the oculogyric centers and pathways. With rare exceptions, simultaneous cephalogyric and oculogyric movements are nearly constant, and Grasset's idea can be enlarged by stating that "the left cortex carries the head and both eyes to the right and vice versa." It is readily seen that a lesion affecting both the oculgyric and and the cephalogyric centers of the same hemisphere will induce a simultaneous paralysis of these two mechanisms, i. e., lateral conjugate deviation of the head and eyes.

REPORT OF CASE

Clinical History.—C. F., a man, aged 51, married, a salesman, was admitted to the Montefiore Hospital on Dec. 2, 1928, complaining of spells of dizziness, movements of the eyes to the right, numbness of the right side of the body, nausea, loss in weight, headaches and occasional sensations of pulsations in the right ear. About fifteen years before, the patient had noticed a dull supra-orbital headache, which recurred at monthly intervals and lasted from one to seven days. Recently, this paroxysmal pain had occurred more frequently, at times lasting for a month; it was noticed only at the end of the dizzy spells. In October, 1928, the patient had attacks of dimness of vision followed by deviation of the eyes to the right and accompanied by a feeling of numbness over the entire right half of the body; it ended with a crying spell. These attacks came at varying intervals; they were not preceded by auras nor accompanied by loss of consciousness, incontinence, biting of the tongue or frothing at the mouth. During the seizure the patient usually called some one, following which he would burst into uncontrollable weeping; he experienced weakness for a time afterward. A marked increase in blood pressure was observed during the attack, a rise from forty to fifty points in systolic pressure. The duration of the attacks varied, lasting from two to fifteen minutes and occasionally for an hour or more, during which repeated seizures occurred. All of the attacks were inferred by the fact that he was awakened by a feeling of numbness over the right side of the body. Many of the seizures consisted solely of slight vertigo, turning of the eyes to the right and inability to turn the eyes toward the left beyond the median line. For the past few years the patient had complained of dyspnea and precordial pain, which were relieved by belching.

He said that he had had no venereal infection. Diphtheria occurred at the age of 18, but left no residual. Alcohol was used to a moderate degree.

Physical Examination.—The patient was well developed. The skin of the forearms and legs showed areas of a macular eruption, which was not syphilitic in nature. The results of examination of all the organs were negative. The pulse rate was 100; the blood pressure was 140 systolic and 100 diastolic, except during the attacks when the systolic pressure rose from forty to fifty points.

Neurologic Examination.—The mental status of the patient was normal. The patient had good intelligence and was emotionally stable. The right palpebral fissure was slightly greater than the left. The pupils were equal, regular and reacted normally. The fundi showed a fulness of the veins, and a tendency toward arterial spasm was reported by the ophthalmologist. There were slight nystagmoid movements when the eyes deviated to the right.

Observations During a Paroxysm.—An acute paroxysm was observed on Dec. 5, 1928, at 5:30 p. m. Shortly before the attack, the patient complained of dimness of vision and vertigo. There was no flushing or blanching of the face. The eyes were strongly deviated to the extreme right, and the patient was not able to move them to the left in spite of strong efforts. There were slight nystagmoid movements, but no convulsive movements elsewhere. The pupils were

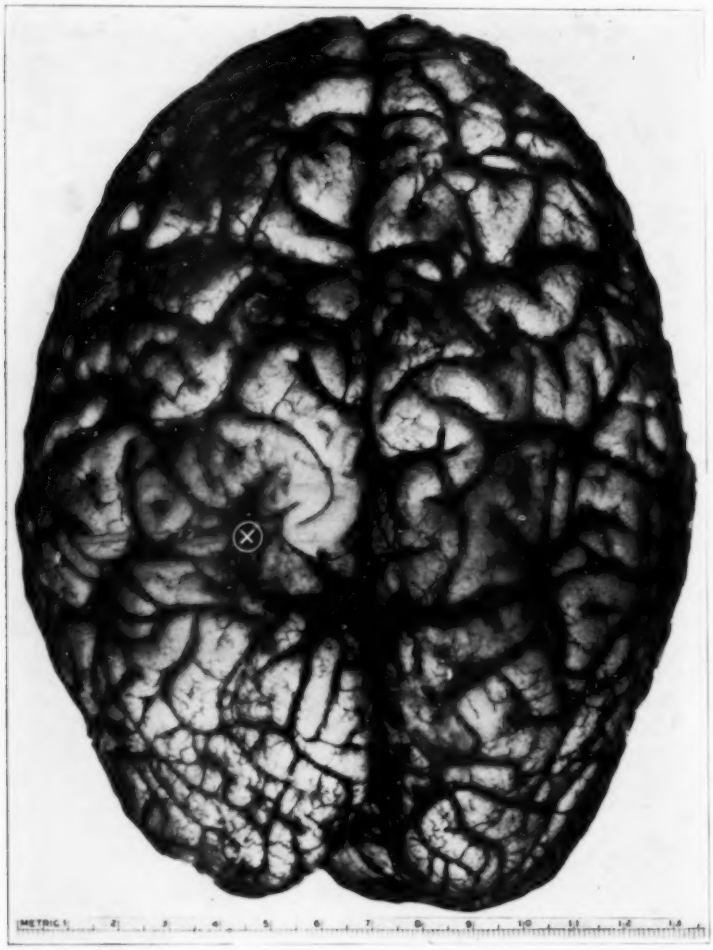

Fig. 2.—Depression of the left parietal area, extending into the postcentral gyrus.

under the influence of a mydriatic and could not be studied, but in a previous seizure the pupils were observed to be dilated and fixed, and the attack lasted over an hour. The patient stated that he could not see objects at his left (this might have been accounted for by the extreme deviation to the right or have been due to hemianopia). During the attack the plantar reflexes were absent; the right

abdominal reflex was not as active as the left; otherwise all motor responses were normal. The oculogyric spasm was immediately followed by convulsive weeping and emotional reaction, as already described. The entire attack lasted about a half minute. Immediately following the attack, the patient complained of dysesthesia, i. e., "pins and needles sensation" over the right side of the body, including the face and occiput. Objectively, there were no sensory changes. The

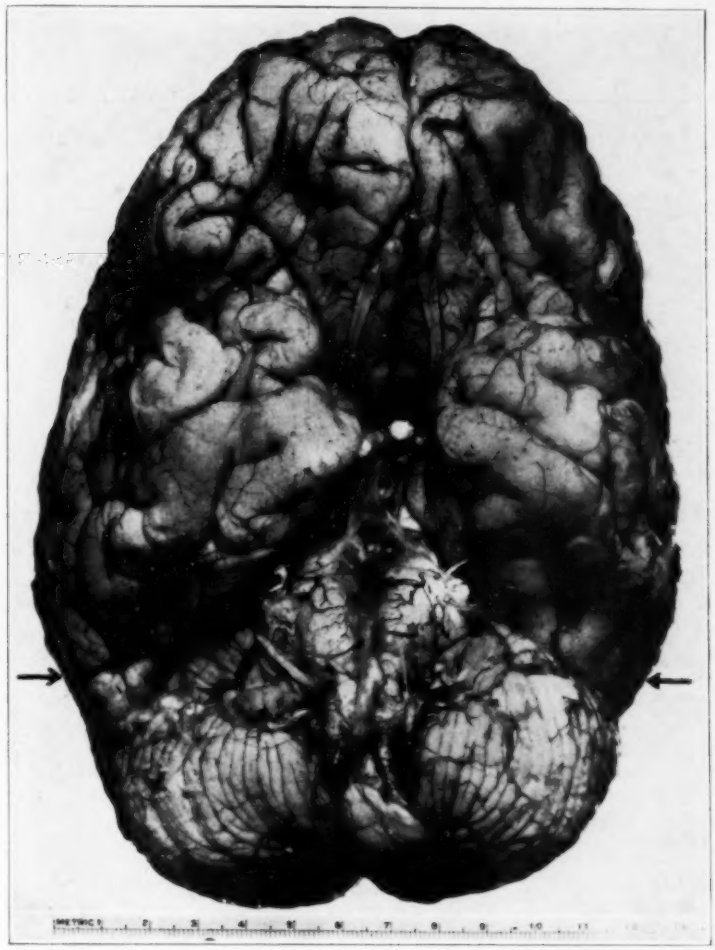

Fig. 3.—Atheromatous plaque at the junction of the two vertebral arteries and along the basilar arteries.

patient complained of dizziness, and on attempting to stand he tended to fall in a forward direction.

Laboratory Data.—Stereoscopic examination of the skull on Dec. 24, 1928, failed to reveal any gross changes in the bones of the calvarium. There was a large, irregular, calcified deposit, which was thought to be situated in the pituitary fossa, as well as calcification of the pineal gland.

Examination of the blood showed: red cells, 4,120,000; white cells, 8,200; differential count, normal. Repeated Wassermann tests of the blood and spinal fluid and the blood chemistry tests were negative.

Course.—The patient was discharged on Dec. 9, 1928, but remained under observation by one of us. Subsequent examinations revealed some limitation in the field of vision of the left eye. An examination of the eyes on Dec. 24, 1928, by Dr. C. E. McDannald showed vision in the right eye 20/65 and in the left, 20/100, which was unimproved by glasses. There was a rotary nystagmus on looking to the extreme left and lateral nystagmus when the eyes were directed

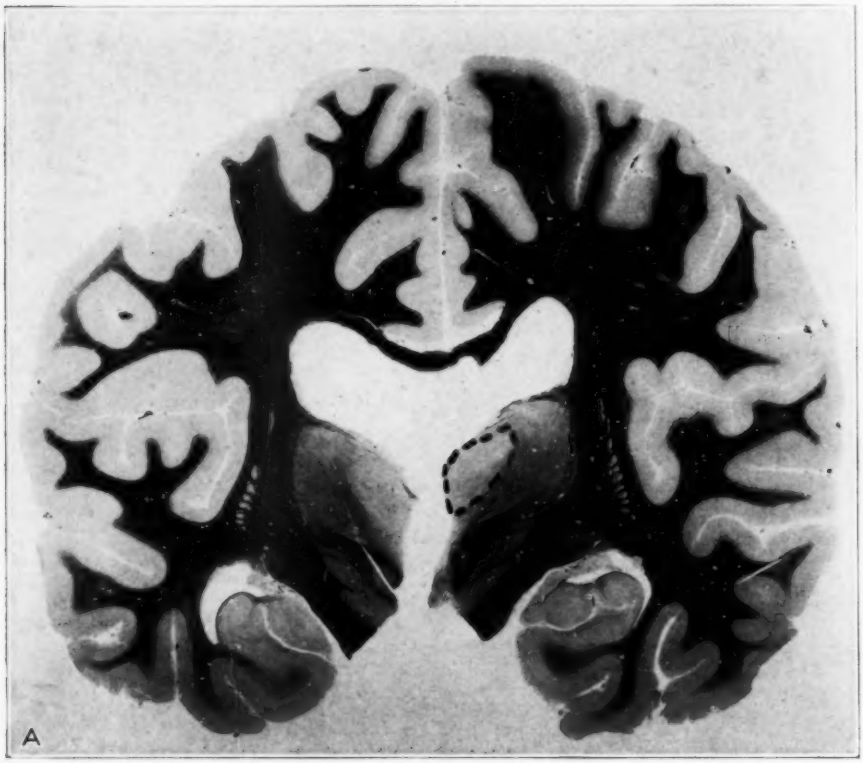

Fig. 4 *A.*—A vertical section of the brain showing a slight area of softening in the medial nucleus of the right thalamus. Modified Loyez (Weil) stain; × 1.

to the extreme right. The pupillary reflexes were normal. The retinal veins were slightly engorged, and the arteries were markedly sclerosed. Field plotting showed contraction for blue, red and green, with a left homonymous, incomplete quadrant hemaniopia below and normal blind spots. A lesion involving the right upper lip of the calcarine fissure was considered.

On January 12, following one of the attacks complete loss of power of the upper and lower extremities, weakness of the right lower facial, deviation of the tongue to the left, thickness of speech, hyperactive reflexes on the right, absent right abdominal reflexes and ankle clonus on the right developed. There were

no sensory disturbances. There was profuse perspiration on the right side of the body. The blood pressure was: left, 140 systolic and 90 diastolic; right, 150 systolic and 90 diastolic. The patient became unconscious and died at home on Jan. 13, 1929. Permission for the removal of the brain only was given, and this organ was removed six hours after death.

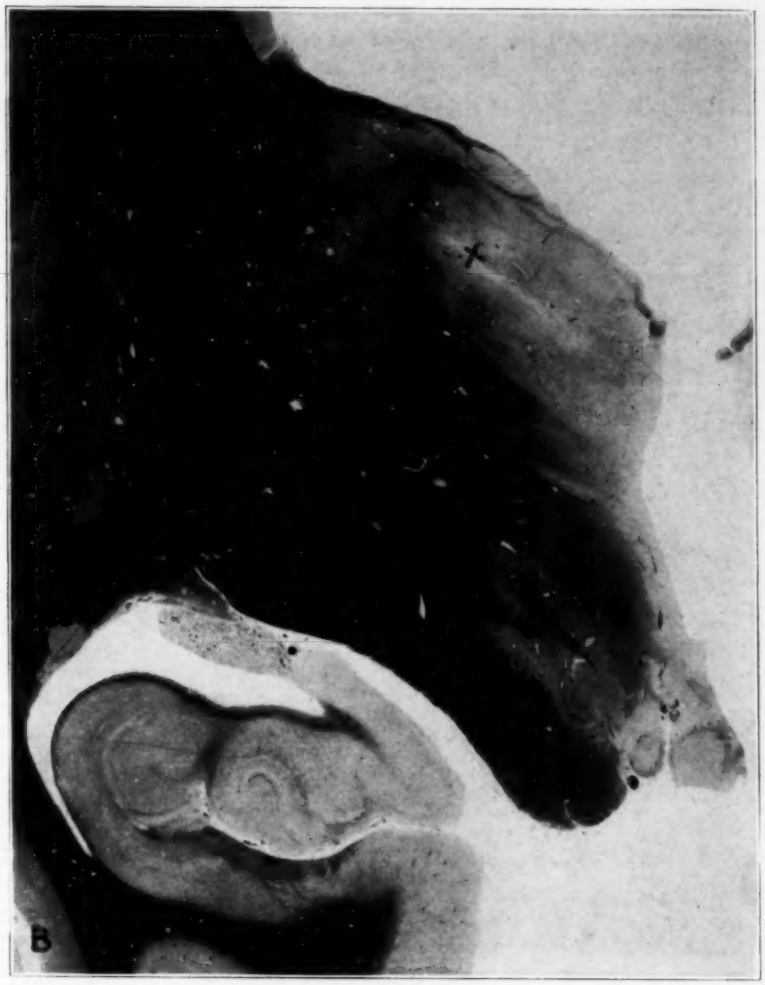

Fig. 4 *B*.—A vertical section of the diencephalon showing area of softening in the medial nucleus of the right thalamus. Modified Loyez (Weil) stain; × 8.

The clinical diagnosis was that of neoplasm or arteriosclerotic process, either in the foot of the second left frontal convolution or in the pons, and involvement of the right calcarine area. Vascular insults were considered most likely.

Autopsy.—Gross Examination: The brain had a normal appearance, with the exception of a depression of the left parietal area (fig. 2) along the left inter-

parietal and parieto-occipital fissures. The convolutions of the occipital region appeared somewhat smaller than normal. The posterior surface of the brain near the junction of the two vertebral arteries revealed an atheromatous plaque completely obliterating the basilar artery at this point (fig. 3). Similar atheromatous changes were found throughout the entire course of the basilar artery and along the posterior communicating arteries. The internal carotids showed moderate arteriosclerotic changes.

The brain was cut in the anteroposterior direction. A small area of softening was found in the medial nucleus of the right thalamus (figs. 4 *A* and 4 *B*). The next section showed an area of softening limited to the cortical area along the left

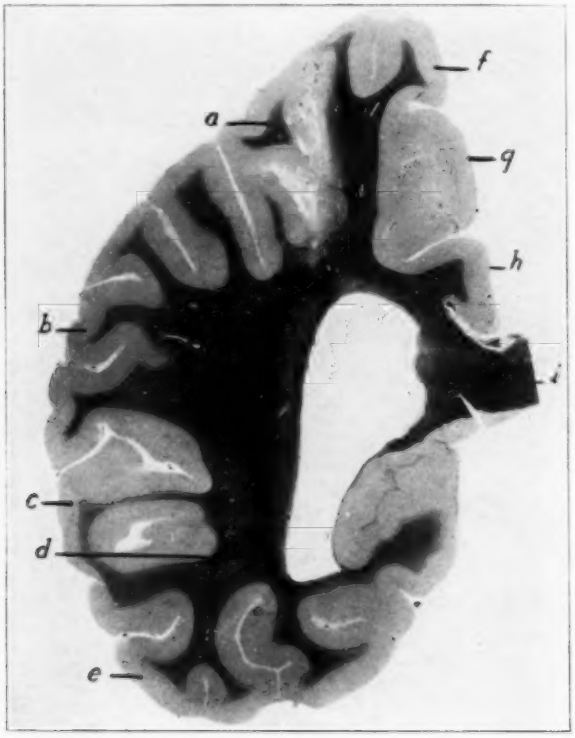

Fig. 5.—Vertical section through the left atrium ventriculi showing area of softening of the left postcentral gyrus. Modified Loyez (Weil) stain; × 3. *a* indicates the gyrus postcentralis; *b*, gyrus temporalis superior; *c*, gyrus temporalis medius; *d*, optic radiation; *e*, gyrus temporali inferior; *f*, gyrus precentralis; *g*, gyrus paracentralis; *h*, gyrus cinguli; *i*, splenium.

postcentral (fig. 5), superior and inferior parietal gyri (fig. 6). The sections through the occipital lobes revealed softening of the left inferior occipital gyrus (fig. 7 *A*) and the right inferior occipital gyrus (fig. 8).

Microscopic Examination: Sections from different parts of the brain, pons, medulla and cerebellum were stained by the Weil, van Gieson, Mallory phosphotungstic, Perdrau, hematoxylin and eosin, and cresyl violet methods.

1. The vertical section passing through the left postcentral gyrus showed, with the Weil stain, demyelinization of some of the white fibers in that region and

partial destruction of the cortex (fig. 5). With higher magnification, this area presented the typical picture of a fresh area of softening, consisting of numerous compound granular corpuscle cells, débris and few sprouting capillaries. There was little tendency toward gliosis. The blood vessels showed moderate thickening of the intima with narrowing of the lumen. The adjacent cortical tissue showed a slight gliosis and destruction, in various stages, of the nerve cells.

2. A section passing through the left occipital horn of the lateral ventricle (fig. 6) and involving the superior parietal lobe as well as the supramarginal gyrus showed, by the Weil method, an area of destruction of the gray and white matter. A slight involvement of the optic radiation was also observed. On

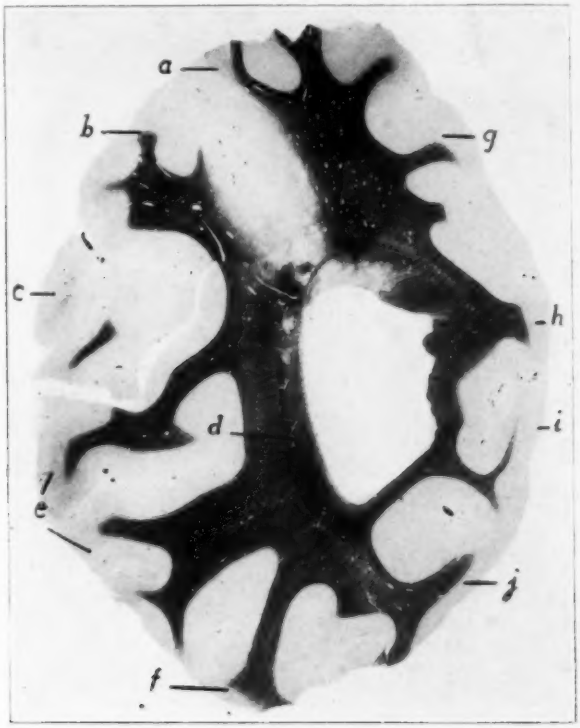

Fig. 6.—Vertical section through the occipital horn of the lateral ventricle showing area of softening of the left superior parietal gyrus and part of optic radiations. *a* indicates the gyrus parietalis superior; *b*, gyrus supramarginalis; *c*, gyrus temporalis inferior; *d*, optic radiation; *e*, gyrus temporalis medius; *f*, gyrus temporalis inferior; *g*, paracentral lobule; *h*, gyrus cinguli; *i*, lingual gyrus; *j*, gyrus temporo-occipitalis.

higher magnification this area of softening had the same histologic appearance as in the previous section.

3. A section passing through the end of the left occipital horn of the lateral ventricle showed an area of destruction involving the white and gray matter of the inferior occipital lobe and the optic radiation, and a slight involvement of the medial occipital lobe (fig. 7*A*). The histopathologic changes were somewhat similar to those in the former sections, with the exception that the vessels in this

area showed more marked arteriosclerotic changes and were more numerous; in the outer border a few were found which showed a collar of compound granular corpuscles simulating perivascular infiltrations. Part of the occipital lobe showed evidences of fresh hemorrhages with extravasation of blood into the nerve tissue (fig. 7 *B*). From the histopathologic observations, this section showed evidence of a very recent process. There was evidence of proliferative endarteritis. The lumina of some of the small vessels and capillaries were completely obliterated.

4. A section passing through the end of the right occipital horn of the lateral ventricle, with the Weil method, showed a partial destruction of the gray and white matter of the right inferior occipital gyrus (fig. 8) and of some of the

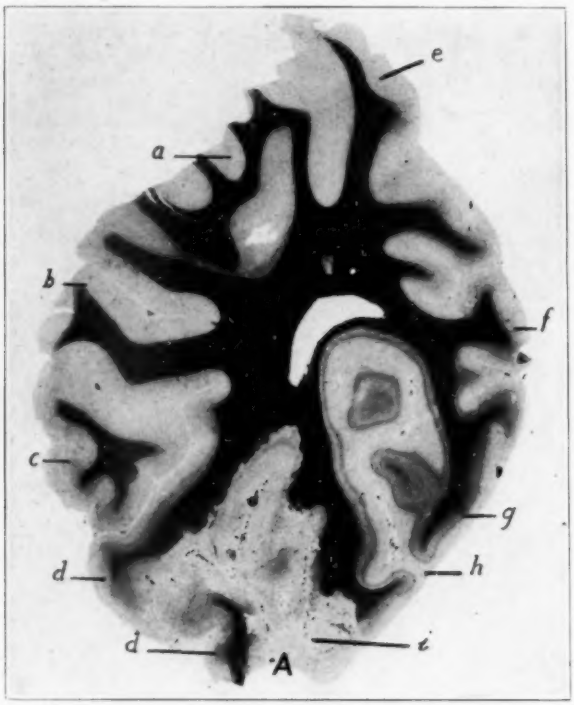

Fig. 7 *A*.—A vertical section through the end of the left occipital horn of the lateral ventricle showing area of softening of the left inferior occipital gyrus. Modified Loyez (Weil) stain; × 3. *a* indicates the gyrus supramarginalis; *b*, gyrus temporalis superior; *c*, gyrus temporalis medius; *d*, gyrus temporalis inferior; *e*, gyrus parietalis superior; *f*, precuneus; *g*, cuneus; *h*, fissura calcarina; *i*, gyrus occipitalis inferior.

fibers of the optic radiation. With higher magnification changes similar to those already described were observed. The lumina of the vessels in the fissure from which this gyrus received its blood supply showed marked narrowing. Some of the smaller vessels were completely obliterated.

5. A section through the diencephalon, by the Weil method, showed a small area of softening involving the medial nucleus of the right thalamus (figs. 4 *A* and 4 *B*). With higher magnification, a definite area of softening filled with gitter cells and blood vessels with thickened intima and narrow lumen was found (fig. 9); the

nerve cells were completely destroyed. The gray matter in the vicinity of this area showed a slight involvement of the nerve cells with slight gliosis at the periphery.

6. Sections in the region of the third, fourth and sixth cranial nerve nuclei, with the Weil method, showed no areas of demyelinization. With the cresyl violet and hematoxylin and eosin methods, these nuclei were found to be intact. The fasciculus longitudinalis posterior throughout its course was normal. Some

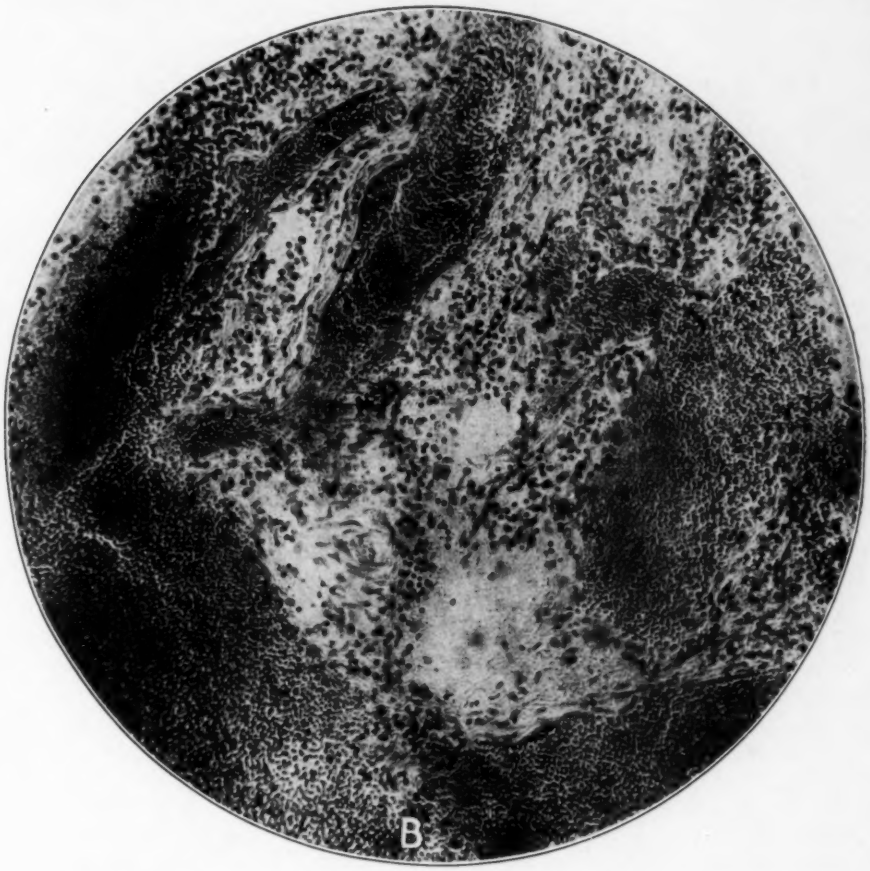

Fig. 7 *B*.—A section with extravasation of blood within the left occipital lobe. Hematoxylin and eosin stain; × 100.

of the pyramidal nerve cells of the pons were shrunken and showed poor Nissl substance. The basilar artery throughout its course showed atheromatous changes with partial obliteration by a partially organized antemortem thrombus (fig. 10).

7. Sections of the medulla oblongata at various levels, with the Weil method, showed a slight paling of the pyramids and the lemnisci. With higher magnification, however, the changes were almost negligible in the form of slight swellings of the myelin sheaths with occasional destruction of the same. With the cellular

and glial stains, this area did not show any of the products of disintegration. The basilar artery at this level showed a partially organized thrombus attached to the intima (fig. 10); the latter was thickened; the elastica interna was broken up. The smaller vessels showed definite thickenings of the intima and media. The obstruction to the circulation was of too recent origin to cause pathologic changes in the bulb.

COMMENT ON CASE

The outstanding clinical features in this case were: oculogyric spasms to the right, unaccompanied by cephalogyric movements, fol-

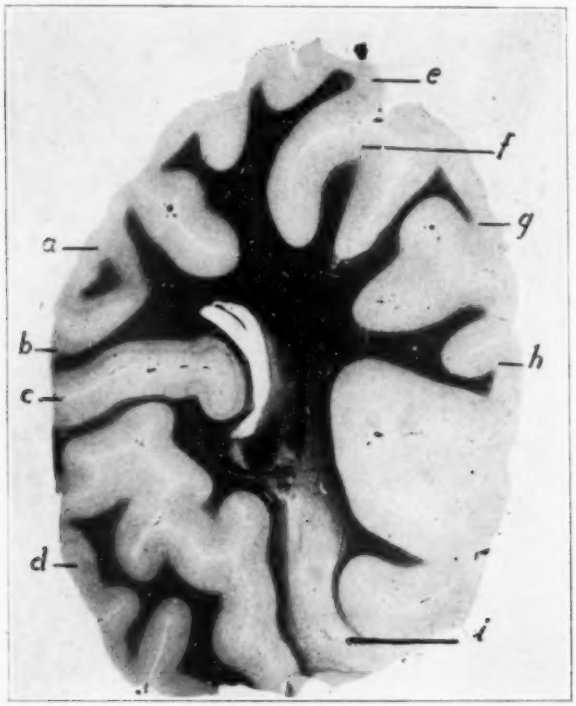

Fig. 8.—Vertical section through the end of the right occipital horn of the lateral ventricle showing area of softening of the right inferior occipital gyrus and demyelinization of the fibers of the optic radiation. Modified Loyez (Weil) stain; × 3. *a* indicates the precuneus; *b*, cuneus; *c*, fissura calcarina; *d*, gyrus temporalis inferior; *e*, gyrus parietalis superior; *f*, gyrus supramarginalis; *g*, gyrus angularis; *h*, gyrus temporalis medius; *i*, gyrus occipitalis inferior.

lowed by a subjective sensation of numbness of the entire right side of the body, emotional outbursts and a left homonymous, incomplete quadrant hemianopia. The obtrusive pathologic lesions were: softening, following thrombosis of the vessels supplying the left postcentral,

superior and inferior parietal, angular and supramarginal gyri, the left and right inferior occipital gyri and a small area of the right medial nucleus of the thalamus.

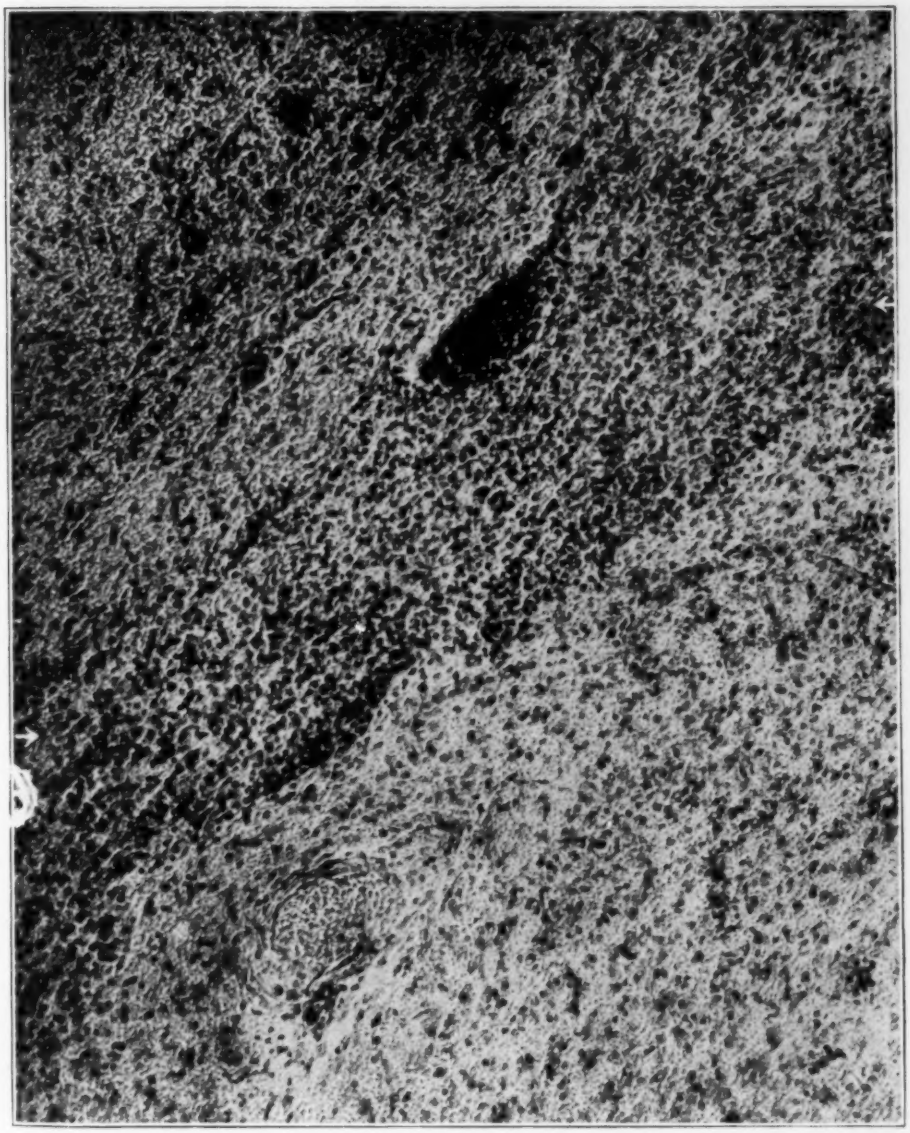

Fig. 9.—Section through the medial nucleus of the right thalamus showing the area of softening filled with compound granular corpuscles. The arrows indicate the areas of compound granular corpuscles. Hematoxlin and eosin stain; × 160.

LOCALIZATION OF LESIONS

Observations on the localization of cerebral lesions in lateral conjugate deviation of the eyes were, with the exception of Foville, first made by Prevost [4] in 1868. Foville,[5] in 1859, mentioned clinical paralysis of associated movements of the eyes to the left in a case of left facial palsy and right hemiplegia. Prevost,[4] experimenting on dogs by pro-

Fig. 10.—Basilar artery showing endarteritis with a thrombus attached to it. Van Gieson stain; × 40.

ducing lesions of the corpus striatum and thalamus, induced conjugate deviation of the head and eyes. He concluded that most of the cases presenting lateral conjugate deviation of the head and eyes were due

4. Prevost, J. L.: De la déviation conjugée des yeux et de la rotation de la tête dans certains cas d'hémiplégie, Thèse de Paris, Paris, Victor Masson, 1868.

5. Foville: Note sur une paralysie peu connue de certains muscles de l'oeil, et sur la liaison avec quelques points de l'anatomie et la physiologie de la protubérance annulaire, Gaz. hebd. de méd. **4**:10, 1859.

to deep-seated lesions near the corpus striatum and the cerebral peduncles. In superficial lesions of the cortex this phenomenon was rarely observed. In one case, by obliterating one of the vessels of the circle of Willis (mention of the exact vessel is not made), a lateral conjugate deviation of the head and eyes toward the side of the obliteration and a hemiplegia on the opposite side were produced without any evidence of softening at autopsy. Death in this case came too soon for the appearance of any marked changes in the nerve tissue. He con-

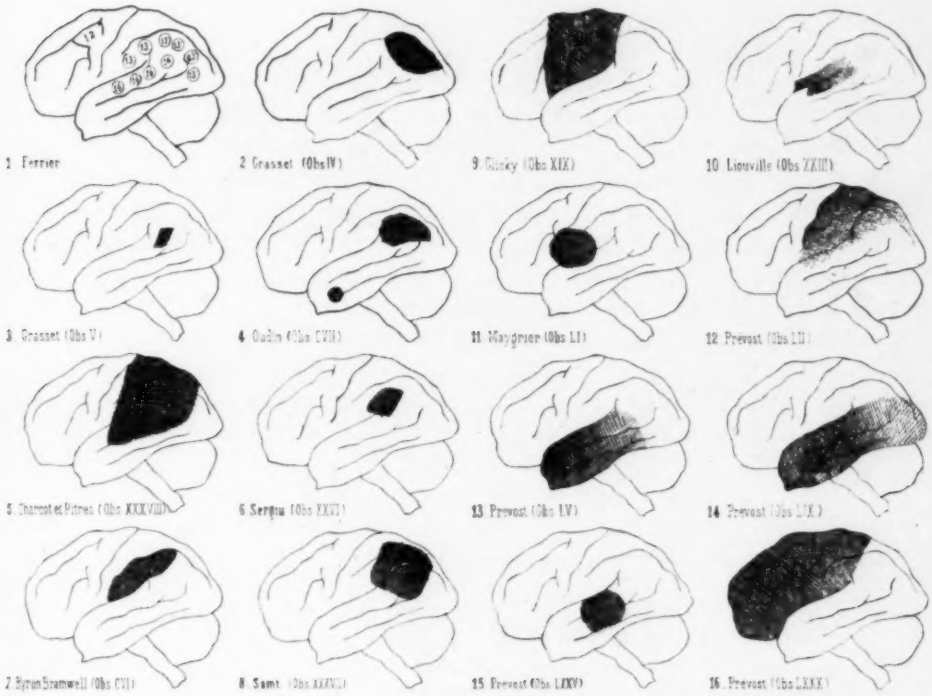

Fig. 11.—Diagram showing involved areas which caused lateral conjugate deviation of the eyes in a series of cases collected from the literature by Grasset, as well as in our case, showing areas in the parietal region stimulated by Ferrier and inducing oculogyric phenomena.

cluded that the cerebral ischemia following the obliteration of the vessels caused the oculogyric and cephalogyric phenomena. Prevost's ideas in regard to localization were inconclusive.

Ferrier,[6] in 1879, on stimulating the base of the first frontal and part of the second frontal convolutions of the brain of monkeys induced

6. Ferrier, David: Localization of Cerebral Disease, New York, G. P. Putnam Sons, 1879, p. 58; The Functions of the Brain, New York, G. P. Putnam Sons, 1886, p. 242.

elevation of the eyelids, dilatation of the pupil, conjugate deviation of the eyes, and turning of the head to the opposite side. Later, he obtained somewhat similar results by stimulating the angular gyrus and the temporosphenoidal convolutions. Landouzy,[7] in 1879, following the experiments of Hitzig and Ferrier, thought that these lesions were located in the right frontoparietal region and differentiated between paralytic and irritative lesions. Grasset,[3] at about the same time, concluded that in conjugate deviation of cortical origin the changes are most likely to be found in the convolutions close to the sylvian fissure and in the inferior parietal and supramarginal gyri (fig. 11). He agreed with other observers that involvement of the fibers from these structures going to the peduncles will give rise to the same disturbance, and on the basis of a number of cases from the literature placed the lesions in the cerebral cortex as illustrated in figure 11. In most of these cases the lesions were not located at the foot of the second frontal convolution, but in the parietal areas and in the convolutions near the sylvian fissure. Landouzy's differentiation into paralytic and irritative lesions was confirmed by Grasset. Wernicke [8] and Henschen [9] placed the lesions in the inferior parietal and supramarginal gyrus. Horsley and Schafer [10] and Beevor and Horsley [11] agreed with the observations of Ferrier.[6] Horsley diagnosed a lesion of the frontal lobe during the beginning of an attack of jacksonian epilepsy accompanied by conjugate deviation of the head and eyes to the opposite side. Mills,[12] during an operation for a brain tumor, by stimulating the posterior part of the second frontal convolution induced complete deviation of the head and eyes to the opposite side. Conjugate deviations of the head and eyes were observed by the same author in a case of tumor of the pons.

7. Landouzy, L.: De la déviation conjugée des yeux et de la rotation de la tête par excitation ou paralysies des sixième et onzième paires, V. A. Delahaye & Cie., 1879.

8. Wernicke: Herderkrankung des unteren Scheitelläpchens, Arch. f. Psychiat. **20**:244, 1889; On Paralysis of Latero-Version, Arch. Ophth. **32**:163, 1903.

9. Henschen, F.: Ueber Geschwülste der hinteren Schädelgrube, Klinische und anatomische Studien, Jena, Gustav Fischer, 1910.

10. Horsley and Schafer: A Record of Experiments upon the Functions of the Cerebral Cortex, Phil. Tr. Roy. Soc. London **179**:1-45, 1888.

11. Beevor and Horsley: An Experimental Investigation into the Arrangement of the Excitable Fibres of the Internal Capsule of the Bonnet Monkey, Phil. Tr. Roy. Soc. London **181**:49, 1890; A Further Minute Analysis by Electric Stimulation of the So-called Motor Region of the Cortex Cerebri in the Monkey, Phil. Tr. Roy. Soc. London **179**:205, 1888.

12. Mills, C. K.: Two Cases of Diseases of the Brain, Brain **2**:547, 1879; Tumor of the Pons Varolii with Conjugate Deviation of the Eyes and Rotation of the Head, J. Nerv. & Ment. Dis. **6**:470, 1881.

Schaffer [13] described a case of cerebral syphilis with a severe lesion of the right frontal lobe and paralysis of associated movement of the eyeballs to the left.

Mott and Schafer,[14] by stimulation of the middle portion of the foot of the second frontal convolution, demonstrated lateral movements of the eyeballs; stimulation of the superior portion of the second frontal convolution caused lateral and downward movements of the eyeballs; of the inferior portion, lateral and upward movements of the eyeballs.

Grunbaum and Sherrington,[15] and Sherrington [16] were the first to use unipolar faradization. The inferior and middle frontal convolutions caused conjugate deviation of the eyeballs to the opposite side. According to them, the frontal area presents "marked differences of reaction from the motor area in the Rolandic region, and it seems necessary to put it in a separate physiologic category. Spatially it is separated from the Rolandic motor area by a field of unexcitable cortex." Ocular movements by faradization of the extreme posterior apex of the occipital lobe, especially the area of the calcarine fissure, were not easily induced.

Long before the investigations mentioned, Schafer [17] demonstrated oculogyric movements by electrical stimulation of the occipital lobes. Bárány, and Cécile and Oskar Vogt [18] found differences from faradization of the foot of the second frontal convolution from that of the calcarine area. In the former the movements of the eyes occurred sooner and lasted much longer than in the latter. Dejerine and Roussy [19] reported conjugate deviation of the eyes in a case of congenital blindness

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13. Schaffer, Karl: Ein Fall von ausgedehenter Meningitis syphilitica der Hirnconvexität und Basis, Neurol. Centralbl. **23**:1035 (Nov. 16) 1904.

14. Mott and Schafer: On Associate Eye Movements Produced by Cortical Faradization of the Monkey's Brain, Brain **13**:165, 1890,

15. Grunbaum, A. S. F., and Sherrington, C. S.: Observations on the Physiology of the Cerebral Cortex of Some Higher Apes, Proc. Roy. Soc. London **69**:206, 1901-1902.

16. Sherrington, C. S.: Integrative Action of the Nervous System, New Haven, Yale University Press, 1911, pp. 279-281.

17. Schafer: On Electrical Excitation of the Occipital Lobe and Adjacent Parts of the Monkey's Brain, Proc. Roy. Soc. London **43**:408, 1888; A Comparison of the Latency Period of the Ocular Muscles on Excitation of the Frontal and Occipito-Temporal Region of the Brain, ibid. **43**:411, 1888; Experiments on the Electrical Excitation of the Visual Area of the Cerebral Cortex in the Monkey's Brain, Brain **11**:1, 1889.

18. Bárány, R.; Vogt, C., and Vogt, O.: Zur reizphysiologischen Analyse der corticale Augenbewegungen, Jahrb. f. Psychiat. u. Neurol. **30**:87, 1923.

19. Dejerine and Roussy: Un cas d'hémiplégie avec déviation conjugée de la tête et des yeux chez un aveugle de naissance, Rev. neurol. (Feb. 15) 1905, p. 161.

in which there was an occipital lesion. Heitz and Bender [20] stand alone in their claim to have seen a case of softening of the posterior part of the temporal lobe that caused lateral conjugate deviation of the head and eyes.

From a study of the clinical and experimental data available, it is difficult to attribute to a definite area of the cortex the localizing function of oculogyric and cephalogyric spasms or paralysis. Experimental data seem to confirm clinical experience in recognizing the foot of the second frontal convolution as the motor "center" for these movements and the most common seat of lesions. Next in order of frequency for the site of oculogyric and cephalogyric disturbances should be considered the inferior parietal and supramarginal gyri, and the occipital area in the region of the calcarine fissure, the last mentioned being the least frequent. Lesions in the course of the fibers originating from these areas may induce lateral conjugate deviation of the head or eyes or both, depending on whether one or both groups of fibers are involved.

The localization of lesions which may interfere with lateral conjugate deviation of the eyes can therefore be grouped as follows:

1. Cerebral Lesions: (*a*) Cortical: A lesion of the anterior center (foot of second frontal convolution, inferior parietal and supramarginal gyri) will induce conjugate deviation of the head and eyes. If the oculogyric fibers alone are involved, there will be no turning of the head. These centers being in close proximity to the center for movement of the extremities, hemiplegia or monoplegia is a frequent concomitant.

According to Landouzy, Grasset and others, if the lesion is paralytic, the eyes will deviate toward the side of the lesion and away from the paralyzed extremities. If the lesion is irritative, the opposite will take place: namely, the eyes will look away from the side of the lesion and will be turned toward the convulsed extremities. A paralytic lesion of the posterior center, that is, the occipital area about the calcarine fissure, will produce deviation of the eyes and head to the side of the lesion and hemianopia on the opposite side. The turning of the eyes in occipital lobe lesions will depend on whether the lesion is paralytic or irritative.

(*b*) Cortical interassociation fibers: Lesions of the association fibers of the occipitofrontal fasciculus which connects the visual cortical area with the frontal centers for ocular muscles, or lesions of other association pathways uniting the cortical eye and head centers may give rise to lateral conjugate deviation. Involvement of these fibers may be the real explanation for oculogyric phenomena in cortical lesions other than those of the foot of the second frontal convolution.

20. Heitz and Bender: Un cas d'épilepsie Jacksonienne débutant par la déviation conjugée de la tête et des yeux avec autopsie, Rev. neurol. **9**:614, 1901.

(*c*) Centrum ovale: As the oculogyric and cephalogyric fibers are close together, lesions of this area will frequently involve both types of fibers with resulting conjugate deviation of the head and eyes. The turning of the head and eyes will be the same as described in lesions of the cortical centers. In exceptional cases the head may turn one way and the eyes the other. Dissociations of this kind cannot be explained except by a lesion destroying the oculogyric fibers and compressing or causing irritation of the cephalogyric fibers or vice versa. Such syndromes were observed by Prevost,[4] Grasset,[21] and Roussy and Gauckler.[22]

(*d*) Internal capsule: As the oculogyric and cephalogyric fibers are close together, an involvement of both fiber tracts, accompanied by hemiplegia and supranuclear facial palsy, is the rule. Occasional lesions of this area may cause conjugate deviation of the head with an associated isolated supranuclear facial without a hemiplegia.

2. Cerebral Peduncles: A lesion at this level will cause crossed conjugate deviation of the head and eyes and paralysis of the face and extremities. In involvement of the pes pedunculi, a Weber's syndrome will result: paralysis of the extremities and face on one side, and oculomotor palsy on the opposite side without oculogyric palsy, for the oculogyric fibers pass in the dorsal or superior portion of the basis pedunculi.

3. Pons: (*a*) A lesion of the superior portion of the pons will cause conjugate deviation to the side of the lesion with a hemiplegia of the face and extremities on the opposite side. The clinical aspect is identical with lesions found in the cortical centers, centrum ovale, internal capsule and cerebral peduncles. (*b*) Lesions of the middle portion of the pons will cause an alternating paralysis, that is, oculogyric and cephalogyric palsy on the same side as the lesion with supranuclear facial palsy and hemiplegia on the side opposite to the lesion, the eyes and head turning toward the paralyzed extremities, that is, away from the lesion if the latter is a paralytic one. The reverse will take place in irritative lesions. This seeming variation is due to the crossing of the oculogyric and cephalogyric fibers. This symptom-complex Grasset called "syndrome of Foville." (*c*) Lesions of the inferior portion of the pons cause lateral conjugate deviation and hemiplegia opposite to the lesion and a facial palsy on the side of the lesion, if the lesion is a paralytic one. The reason for this is that at this level

21. Grasset, J.: De la déviation au sens opposé de la tête et des yeux, Semaine méd. (May 18) 1904.

22. Roussy and Gauckler: Un cas de déviation en sense opposé de la tête et des yeux, Rev. neurol. (July 30) 1904, p. 763.

the oculogyric and cephalogyric and facial fibers have decussated; the pyramidal tracts have not.

Lesions involving the nucleus of the sixth nerve alone will cause only paralysis of lateral movement on the same side as the lesion, with conjugate deviation toward the same side. Owing to the close proximity of the genu of the facial nerve as it turns round the abducens nucleus, involvement of the sixth nucleus will usually also cause a peripheral type of facial paralysis.

4. Posterior Longitudinal Bundle: A lesion of these fibers in the region of the oculomotor nuclei will cause lateral conjugate deviation with a facial palsy, for the fibers of this nerve are in close proximity to the posterior longitudinal bundle.

5. Deiters' Nucleus and Its Connections: Lesions of these structures will produce lateral conjugate deviation of the eyes, but will be accompanied by nystagmus. Vertigo, nausea, anxious states, evanescent auditory phenomena and pain in the region of the trigeminal nerve may also occur in involvement of this pathway.

6. Cerebellum: Lesions of the cerebellum may induce lateral conjugate deviation. These have been demonstrated by Thomas [23] experimentally, but the deviation in these cases is evanescent, lasting at most a few days.

COMMENTS

The clinical picture in our case, with its unusual feature of oculogyric spasm, pointed definitely to a pathologic process in the left cerebral hemisphere. The conspicuous absence of cephalogyric movements was accounted for by lack of involvement of the center for movements of the head. To explain the subjective sensory disturbances accompanying the oculogyric spasm the lesion must be placed either in the left postcentral gyrus or in the pathways carrying this type of sensation. The latter fibers would have to be in proximity to the oculogyric fibers. The emotional outburst immediately following the seizure, which can be classified as a type of "forced crying" not unlike that seen in pseudobulbar palsy, multiple sclerosis and other thalamic lesions, accompanied by the subjective sensory disturbances, suggested the left thalamus as the site of the lesion. Sachs, by stimulating the posterior third of the dorsal half of the lateral nucleus of the thalamus, obtained conjugate deviation of the eyes to the opposite side.

As observed from the pathologic lesions in this case, the foot of the second frontal convolution on either side was free from involvement. Although a thalamic lesion was found, this was located on the right side, and the nucleus involved was the medial nucleus of the thalamus,

23. Thomas, A.: Le cervelet, Paris, G. Steinhal, 1897.

which is said to receive fibers from the olfactory centers, small contingents of the medial lemniscus and spinothalamic tract and to send fibers to the caudate and subthalamic nuclei.

The lack of any pathologic changes, either gross or microscopic, in the foot of the second left frontal convolution or the left lateral nucleus of the thalamus, eliminates these structures as a possible cause for the conjugate lateral deviation observed in this case.

The other area, which occasionally has been described as causing oculogyric and cephalogyric deviation, is the visual area around the calcarine fissure. The bilaterality of involvement of the two inferior occipital gyri would make this localization even more difficult, as the occipital lobe lesion could not account for the peculiar sensory disturbances. However, the partial softening of the right inferior occipital gyrus and of the optic radiation accounts for the left homonymous incomplete quadrant hemianopia.

From the experiments of Ferrier and others by stimulation of the anterior and posterior limb of the angular gyrus, whereby conjugate lateral deviation of the eyes was induced, and from Grasset's collection of cases it has been conclusively shown that most of the lesions were not located in the second frontal convolution but in the parietal lobes, especially the gyri which were in close proximity to the sylvian fissure (fig. 10).

Riley [24] in the report of a case somewhat similar to ours of oculogyric spasm to the right, with the exception of definite and permanent sensory disturbances of the right side of the body, placed the lesion clinically "in the mesial fillet in its internal portion, together with the pes lemniscus superficialis contained within the fillet and the emergent fibers of the oculomotor nerve of the left side as it passes through this part of the mesial fillet."

The descriptions of lesions involving the parietal part of the cortex in oculogyric spasm, the area of softening in this case involving the postcentral gyrus, superior parietal, angular and supramarginal gyri, and the subjective sensory disturbances following the oculogyric spasm induced us to place the lesion for this syndrome in this area. We are at a loss in an attempt to link the emotional outburst to the pathologic observations in this case, unless we regard the entire picture as a jacksonian seizure, in which emotional outbursts following attacks are not uncommon.

As a last possibility in the localization of the lesion in this case, the involvement of the interassociation pathways, especially the occipitofrontal fasciculus which connects the visual area in the cortex with the

24. Riley, H. A.: Conjugate Deviation of the Eyes with the Presentation of a Case, Neurol. Bull. **2**:181 (May) 1919.

frontal centers for ocular muscles, should be considered. Stimulation of these fibers in the vicinity of the inferior parietal gyrus by Ferrier may be the explanation for the success in obtaining lateral conjugate movements of the eyes and head. Lesions of the same fibers may be the explanation of Grasset's collection of cases with latent oculogyric and cephalogyric movements.

The arterial spasms of the retinal vessels, the absence of extensive areas of softening to account for a permanent conjugate deviation of the eyes and the short duration of the oculogyric spasm speak in favor of an irritative lesion, due either to arterial spasm or to irritation of the oculogyric fibers by the products of distintegration from the destroyed areas.

SUMMARY

A case of oculogyric spasm without cephalogyric movement, subjective sensory disturbances on the right and a left homonymous incomplete quadrant hemianopia is presented. Pathologically, there was a multiplicity of lesions of the cortex in the form of small areas of softening of the left postcentral, inferior and superior parietal, angular, supramarginal and bilateral inferior occipital gyri, as well as a small lesion in the medial nucleus of the right thalamus. The main cortical center for movements of the head and eyes, the foot of the second frontal convolution, was free from any involvement. The lesions that can best explain the observations in this case were in the left postcentral, superior and inferior parietal, angular and supramarginal gyri. As it is disputed whether this area is a center for oculogyric and cephalogyric movements, a possibility of involvement of the intercortical association pathways, the occipitofrontal fasciculus, connecting the cortical visual area with the frontal centers for ocular muscles, should be considered.

ABSTRACT OF DISCUSSION

DR. SMITH ELY JELLIFFE: In this article we have an excellent illustration of what has been known for many years, that oculogyric spasms and oculogyric crises occur in a vast variety of diseases other than epidemic encephalitis. It is interesting also that in the history of the recent revival of interest in oculogyric spasms that the first case recorded in the mass of the literature is not a case of encephalitis at all, but is a case by Staehelm reported in the *Schweizerische medizinische Wochenschrift* related to that of Dr. Goodhart and Dr. Davison. What little I have to say concerning these situations concerns itself exclusively with encephalitic cases, from which a few thoughts may be drawn.

The first case is of interest because it is the first case of oculogyric crises observed in encephalitis by Rossi, the Italian observer, in 1922. The eye movements are upward. In most encephalitic cases the ocular movements are upward and to the right, sometimes to the left. It is, moreover, interesting in encephalitic cases to find that the spasms have a tendency toward ambivalence. They may begin down and to the left, and end up and to the right, or they may begin to the right, and end up to the left. The ambivalence is of much interest in diagnosis, as to whether lesions like those described this evening are present, or whether

one has to deal with a tumor or with encephalitis. With oculogyric crises of a paroxysmal nature and showing ambivalence of the motions, the evidence is in favor of postencephalitis.

The second case is also one of the older cases by Paulian, in which one sees the characteristic oculogyric spasm, as well as the cephalogyric spasm; the expression of anxiety is manifest.

The third is a case reported by Ewald. One of the interesting things about it bears on what Dr. Goodhart said of emotional reactions having certain analogies to multiple sclerosis reactions. I hoped that he might have said more about the mental content of the reactions. This patient had very marked reactions, and his mental content chiefly concerned itself with *a, e, i, o, u,* the vowels; then he was concerned with the consonants, which in the emotional reaction were correlated with men and women. He had a mental content which was of interest psychoanalytically.

The fourth case is another one of Ewald's with the same type of anxiety; you see him in the more or less trance state of oculogyric crises; in the next picture the eyes have gone up and to the right, and there is still evidence of a residual negative symptom-palsy in the right eye.

The fifth case is a cinematographic reproduction of one reported by Fischer, and the sixth is one by Marie in which the agonizing character of the whole musculature is evident. When one commences to study the mental content one begins to get an idea of some meaning behind the more particular manifestation in each case.

Finally, there are some cases by Dr. Farnell, in Providence, and some in New York, just to show the symptomatology of the situation in which the paroxysmal nature of the situation is brought to mind.

I shall touch hastily on some things concerning these crises, when they are paroxysmal or more or less continuous. I need not refer to the well known point of view of Hughlings Jackson, first called to our attention concerning negative and positive signs which may be seen in dissolution of the functions of the nervous system. Dr. Goodhart and Dr. Davison have presented interesting slides of what may be called the negative symptoms, that is, the symptoms due to actual destruction of nerve tissue. But the functions of the body as a whole are still operating, in case the patient lives, in spite of these definite lesions. The positive aspect of the symptomatology, particularly in the mental sphere, is of great interest. For that reason it seems that we have to put considerably more emphasis on the interpretation of the functional side of the situations, i. e., the purpose that the body is trying to fulfil in spite of the lesions. One might ask what is the function of these eye movements? What are they doing for the patient? Hence, I raise this question: What is the patient trying to accomplish through these movements? Evidently, he is trying to get away from something. There is great anguish in many. How does the eye attempt to get the patient away from painful situations? That there is a mechanism to bring this about, and that it may be localized in a complex chain of structures is evident, but after all this is said and done, what is the meaning of the whole thing? For has not everything some meaning, if one is only able to find it?

DR. E. D. FRIEDMAN: This case illustrates a point that has been brought out by Foerster in his recent work on the surgical aspects of epilepsy. He has succeeded in mapping out in the cortex of patients with epilepsy, areas which he calls Adversivfelder, stimulation of which gives rise to conjugate deviation of the eyes; one of the important areas is in the parietal lobe. This case supplies suggestive anatomic proof of the existence of such a center in a patient who was carefully observed clinically. One must agree that such a function as conjugate movement of the eyes is a complex phenomenon, and that there must be a number of sources from which it can be elicited.

I shall venture to suggest a teleologic theory in addition to the psychoanalytic mechanisms to which Dr. Jelliffe has referred, namely, that a person would tend to look in the direction of a sensory stimulus. The existence of a center for

conjugate deviation of the eyes, in the parietal lobe — the end-station for sensory impressions — would seem to lend weight to this assumption.

DR. JOSHUA ROSETT: The case presented cannot be depended on for the localization of the oculogyric movements. The lesions were numerous and widespread. Dr. Goodhart's citation of opinions of other investigators who attempted to localize these movements is a little bewildering, since both stimulation as well as destruction of the frontal, the parietal, the occipital and the temporal lobe appear to have produced these movements. How little dependence can be placed on some of these results is perhaps evidenced by the fact that a fronto-occipital fasciculus has been dragged in for the purpose of localizing these movements. I am certain that that fasciculus does not exist. Adolph Meyer, in his anatomic studies of the visual pathways, in 1907 (*Transactions of the Association of American Physicians*), came to the conclusion that there was no such fasciculus. In 1922 (*Brain*), I produced a series of microscopic sections prepared by a new method in such a way that an entire cerebral fiber system could in large part be traced in a single slide, and in which minute bundles of fibers could be followed for several inches. These slides showed definitely that what had been taken for a continuous bundle extending from the frontal to the occipital lobe really consisted of two separate systems. One of these arrived from the frontal and prefrontal regions and, assuming for some distance a horizontal course, bent downward to enter the lateral nucleus of the thalamus; while behind, the fibers were merely those of the tapetum of the corpus callosum. The fronto-occipital fasciculus was discovered by Forel and Onufrowitch. The only evidence which they adduced for its existence was their observation of a gross specimen of the brain of an idiot with a defect of the corpus callosum. It owes its persistence to Dejerine, who, on the strength of evidence just mentioned, committed the error of popularizing it in a hundred pictures in his monumental work on the anatomy of the nervous system.

The case presented appears to me to be one of epilepsy. In this disease, the movement of the eyeball to one side is not at all uncommon. Gowers has given a description of these movements which for its beauty and accuracy may be considered as a classic, and most of us have seen oculogyric movements in similar cases.

DR. I. S. WECHSLER: I wish to ask a question about the localization of the lesion. This question is prompted by the remark of Dr. Jelliffe as to Hughlings Jackson's conception of negative and positive symptomatology. If the patient had a suprasegmental destructive lesion anywhere in the pyramidal pathways before they crossed to the lower nuclei, then of necessity the deviation of the eyes ought to have been to the side of the lesion and not to the opposite side. If you destroy the left motor fibers which move the eyes to the opposite side, the eyes ought to turn back to the left. A left-handed irritative lesion would move the eye to the right. Assuming there was a destructive lesion, the movement was opposite to what one would have expected to be the result. Therefore, one must assume that in this case there was at first an irritative process; it is not necessary to postulate a psychopathologic compulsive mechanism.

DR. S. PHILIP GOODHART: In answer to Dr. Rosett, we stated definitely that the pathologic process left us perplexed as to localization. There was no attempt to make a definite localization to account for the oculogyric crises.

As to the occipitofrontalis fasciculus, I have always assumed it to be there. I have seen the specimens to which Dr. Rosett referred, and I recall that there were a number of variations in his observations from the ordinarily accepted distribution of fibers described by erudite anatomists. It is a question as to who is correct. I rather take it that Dr. Rosett's observations satisfy at least himself. His preparations were excellent technically, and I can only say that they showed divergences from the ordinarily accepted tracts; I am not in a position to say whether the occipitofrontalis fasciculus exists, whether the older men are correct or whether the later observations and the very splendid work of Dr. Rosett are more determinative.

As to epilepsy, I must differ from Dr. Rosett as to this being a case of epilepsy. There is to my mind nothing in the situation which indicated that; the pathologic process would indeed be a revelation for epilepsy. We considered that as a possibility, but I feel satisfied that we are dealing primarily with what has been spoken of as oculogyric spasm, on perhaps a vascular irritative (spasm?) basis, and later occlusion and degeneration of the tissue.

In regard to the irritative phenomena of which Dr. Wechsler spoke, we must have had irritation in the beginning. A study of the several lesions discloses a pathologic process suggesting different periods — acute and more chronic changes.

It is interesting to know that Dr. Agatson at the Montefiore Hospital showed these lesions in the orbital vessels demonstrating arterial spasm. We presented this case not as one definitely determined, but as one of difficult pathology and especially interesting from the clinical standpoint. In my own experience, I have never seen an oculogyric spasm, except in cases of encephalitis, and I have not seen it associated with isolated lesions running over a period of time.

A reference to Grasset's localization chart, as presented by Dr. Davison, indicates how varied in site may be lesions affecting oculogyric tracts.

DR. DAVISON: In answer to Dr. Rosett, from complete serial sections in three dimensions prepared by Dejerine and a similar series prepared by us at the Montefiore Hospital, to our satisfaction, we believe that the occipitofrontalis fasciculus exists. Furthermore, we are not placing the lesion in this fasciculus, but are offering it as a possibility.

SYPHILIS (?) OF THE OCULOMOTOR NERVE*

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CHICAGO

An isolated paralysis of the third (oculomotor) nerve may be caused by a number of morbid conditions. Of these may be mentioned syphilitic basilar meningitis, tumors, aneurysms of the arteries of the circle of Willis, lesions of the cavernous sinus or of any other structure at the base of the brain adjacent to the third nerve. In the case here recorded none of these etiologic factors was present. The cause of the paralysis lay in a disease of the nerve itself and was detected only after a thorough microscopic study.

REPORT OF A CASE

Clinical History.—A widow, aged 58, entered my service at the Cook County Hospital on Feb. 13, 1928, because of severe headaches, pain in the neck and inability to open the right eye. She stated that aside from having a high blood pressure she had never been seriously ill; she had never had any infectious or contagious disease. She coughed occasionally, but said that there were no pains in the chest, hemoptysis, night sweats or similar troubles. For the last ten months she had been suffering from headaches, palpitation and "anemia," for which she had been receiving "horse serum injections." The present trouble, which was of only five days' duration, began suddenly with a severe stabbing pain in the head, neck and the right side of the face. She became dizzy but did not fall and was able to go to bed and call for help.

On examination the patient was somnolent and appeared markedly anemic. Occasionally she would become restless and moan because of severe pain in the head. The right upper lid drooped; the right pupil was larger than the left, but was round and regular and reacted sluggishly to light and in accommodation. The right eyeball failed to move upward, inward or downward, while the external or lateral movements were performed freely. The rest of the cranial nerves were normal. The fundus of the right eye was hyperemic and blurred; the left fundus could not be examined because of a cataract in that eye. The neck was somewhat rigid, with a positive Kernig and a doubtful Brudzinsky sign. Signs of paralysis or paresis in the extremities were absent. The muscle power was good; the tendon reflexes were normal in the upper extremities, and in the lower extremities they were obtained with reenforcement. The Babinski and Chaddock

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* From the Pathologic Laboratories of the Research and Educational Hospitals of the University of Illinois and the State Psychopathic Institute.

signs were noted bilaterally at the first examination, but were entirely absent on subsequent examinations. Genito-urinary and trophic disturbances were absent. The corneal and conjunctival reflexes were normal; the gait and the Romberg sign could not be tested, as the patient was too ill to leave bed.

An examination of the chest revealed a few moist râles in the lower lobes of both lungs and along the axillary lines, and soft blowing murmurs at the apex of the heart which was enlarged to the left. The abdominal organs were normal.

Laboratory Examinations.—The spinal fluid was bloody, and under a high pressure, which could be increased by pressure on the jugular veins (positive

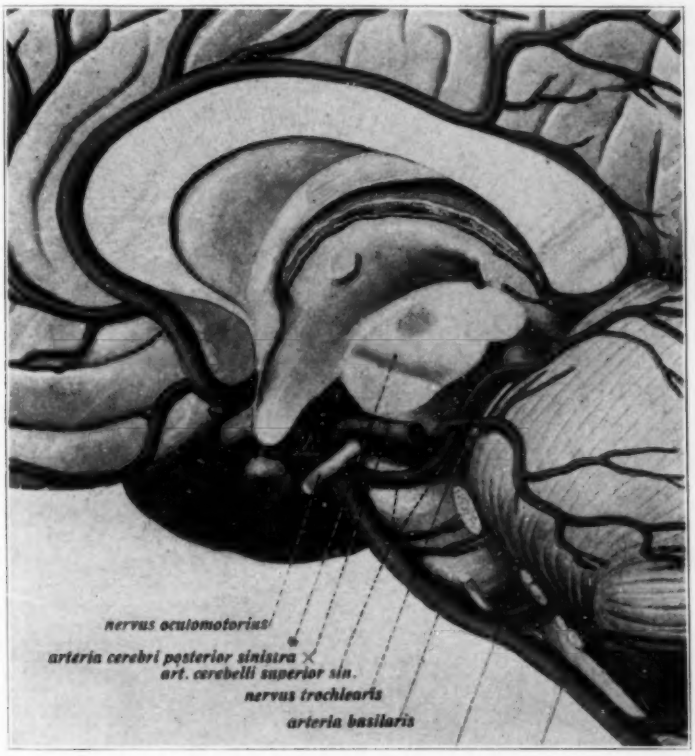

Fig. 1.—The third nerve lies between the arteria cerebri posterior and the arteria cerebelli anterior, close to the posterior communicating artery (from Sobotta's Anatomy).

Queckenstedt sign); the Wassermann test was negative with both the blood and the spinal fluid. The blood pressure was 226 systolic and 96 diastolic; the temperature was 98.8 F.; the pulse rate was 80 and the respiration rate was 16. Chemical examination of the blood revealed: urea nitrogen, 7.47 and 12.2; urea, 1.50; creatinine, 1.35, and sugar, 76 and 130 mg. The urine contained albumin (4+) and a few epithelial cells.

Roentgen examination showed normal sinuses and an exceptionally small sella turcica.

A provisional diagnosis of a basilar meningitis (tuberculous or syphilitic?) was made, and the patient was given antisyphilitic treatment (mercury inunctions).

Course.—The headaches subsided, and the somnolence and the meningeal symptoms (rigidity of the neck and the Kernig sign) disappeared. The tendon reflexes became normal throughout, and though the oculomotor nerve paralysis remained stationary, the patient became cheerful and cooperative, and appetite and sleep improved greatly. Another spinal puncture, performed on Feb. 17, 1928, again yielded a bloody spinal fluid under increased pressure with a negative Wassermann test.

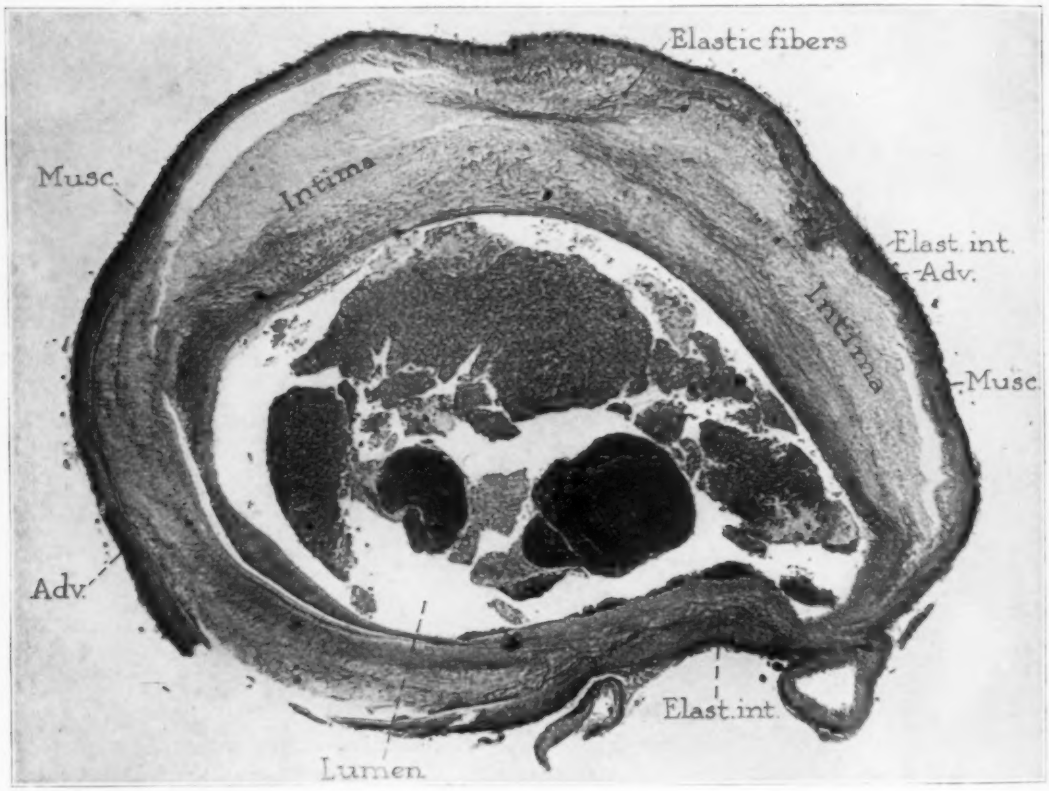

Fig. 2.—Posterior cerebral artery; a transverse section. Van Gieson stain.

Examination of the blood on this date revealed 4,000,000 red cells, 7,000 white cells and 80 per cent hemoglobin. The blood pressure at this time was 250 systolic and 100 diastolic.

The persistent bloody spinal fluid, the high blood pressure and the failure of the oculomotor paralysis to yield to antisyphilitic treatment suggested a vascular lesion at the base of the brain, probably an aneurysm. It was thought that the pressure by the latter on the nerve was responsible for its paralysis, and that probably a leakage from the aneurysm was the cause of the hemorrhagic spinal fluid.

On March 3, 1928, the patient began to show increased temperature, especially during the afternoons; she was often restless and weak and at times irrational. The condition grew progressively worse, and the patient died on June 12, 1928.

Necropsy.—Postmortem examination by Dr. R. Jaffé and Dr. R. Lifvendahl revealed among other conditions: acute miliary tuberculosis with involvement of the lungs, liver, spleen and kidneys; caseation of the tracheobronchial lymph glands, with softening and cavitation; fibrocaseous tuberculosis in the apical

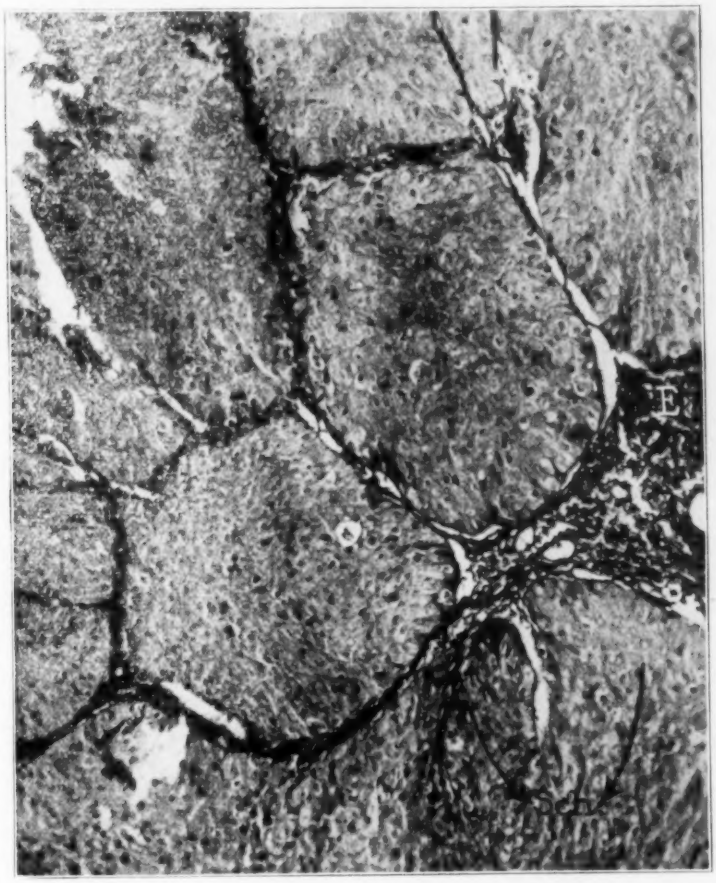

Fig. 3.—The nerve is divided by the endoneural membranes into bundles; at *Sch*, proliferated Schwann cells replaced a nerve bundle; at *E*, the endoneurium replaced the parenchyma of the nerve; a higher magnification is given in figure 5. Bielschowsky counterstained with Alzheimer-Mann; × 135.

median portion of the lower lobe of the left lung; syphilitic aortitis and a slight atheromatosis; marked arteriosclerosis of the medium-sized arteries of the heart and brain; arteriosclerosis of the kidneys; inequality of the pupils; moderate emaciation, dehydration and anemia.

A macroscopic examination of the brain revealed no hemorrhages, aneurysms, tumors or softenings. The meninges were transparent and not thickened; they

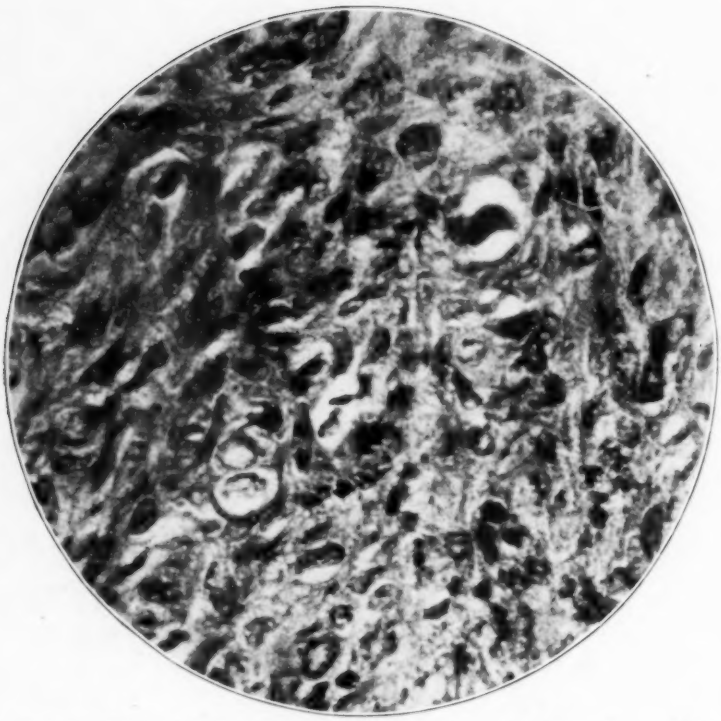

Fig. 4.—Foci of Schwann cells. Same stain as in figure 3; × 500.

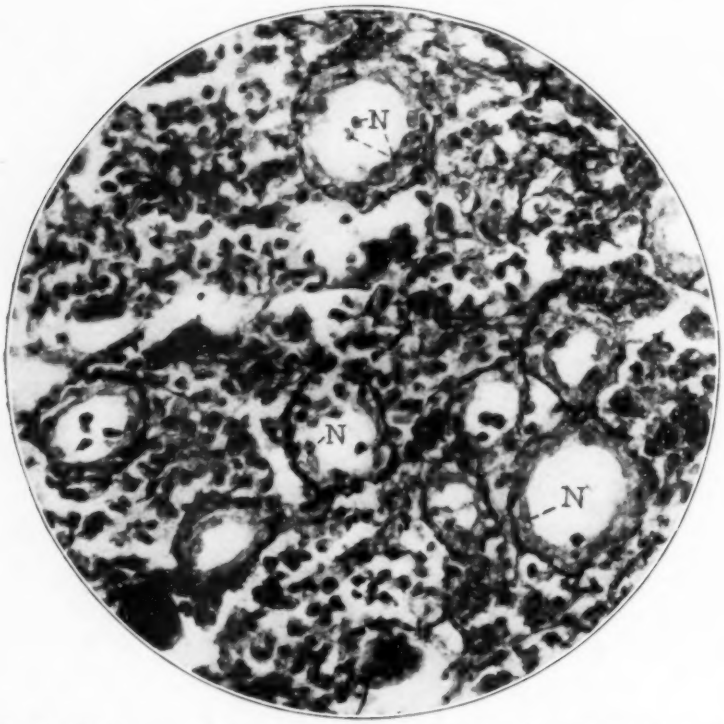

Fig. 5.—High power view of an infiltrated hyperplastic endoneural membrane. The loops (black rings) contain remnants of nerve tissue (*N.N.N.*) plastering their inner surface. Same stain as in figure 3; × 350.

were not adherent to the brain tissues. The cerebral ventricles were of normal size. The basilar arteries were patent; their walls were thickened and the posterior cerebral arteries were in close contact (fig. 1) with the third nerves, especially on the right, where the nerve appeared compressed and flattened. The topographic relationship between the sclerosed blood vessel and the adjacent nerve is shown in figure 1, reproduced from Sobotta's textbook of anatomy.

A microscopic examination revealed changes in the blood vessels and in the right oculomotor nerve. The vascular changes are shown in figure 2. The lumen of the artery (right posterior cerebral) was patent and was lined by endothelial cells, which covered an hypertrophied and proliferated intima. In some places

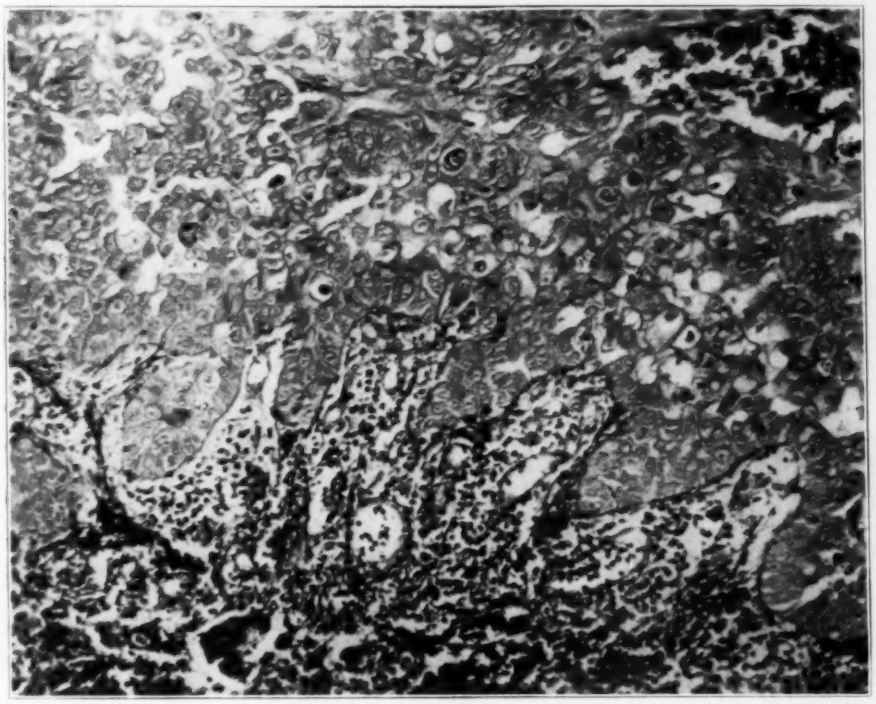

Fig. 6.—The nerve (above) is invaded digit-like by the proliferated and infiltrated endoneurium; the infiltrated masses show loops of connective tissue forming vacuoles (pictured under higher power in figure 5). Same stain as in figure 3; × 140.

this was covered by numerous fibers and fibrils of the broken up internal elastic and connective tissue, with large masses of lipoids. A narrow strip of the muscular tunic lay next to the intima from which it was separated by the internal elastic membrane. The adventitia appeared as a sclerosed band, poor in nuclei, and showed no infiltrations with lymphocytes, plasma cells or other elements. The vascular changes may be classified as proliferation and subsequent degeneration of the intima and the inner elastic membrane.

The changes in the oculomotor nerve were vascular and parenchymatous. Its architecture was generally preserved. The nerve consisted (fig. 3) of smaller and

larger fasciculi or funiculi, each of which was surrounded by an endoneural connective tissue membrane. The nerve fibers in many fasciculi were preserved, but their myelin was tumefied and fenestrated (artefact); the axons were usually disfigured or absent, and the Schwann membrane often appeared hypertrophied. In some funiculi the nerve fibers could not be made out. They had been replaced by islands of hypertrophied and proliferated Schwann nuclei, rich in chromatin (figs. 3 and 4), and often contained abundant cytoplasm. Many such cytoplasmic Schwann cells were rich in fibers. In some funiculi it was possible to note the breaking up of the myelin and axon, the presence of spirals of nerve fibers and their fragments with the formation of so-called ellipsoids. Some Schwann

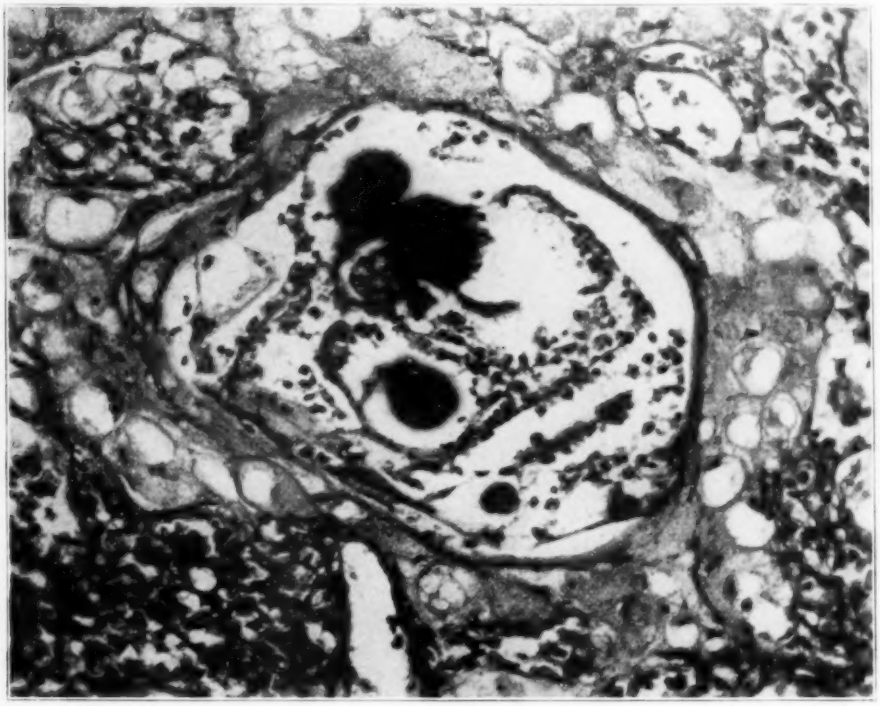

Fig. 7.—A vacuole containing remnants of nerve tissue and bordered by a connective tissue strand. Same stain as in figure 3; × 300.

cells contained large vacuoles harboring minute fragments of myelin or axon. The mesenchymal changes were much more marked. The endoneural membranes that enveloped single funiculi were often hyperplastic; their meshes were distended and densely packed with hematogenous elements (fig. 5)—lymphocytes and especially plasma cells. Numerous and dense as were the cellular infiltrations, they did not form gummas; they did not obstruct the lumens of the blood vessels, nor were the adventitial spaces of the latter excessively infiltrated. The infiltrated endoneurium invaded, digit-like, the parenchyma (fig. 6), which in some areas was transformed into a mass of connective tissue strands. These formed numerous loops which on superficial examination somewhat resembled blood vessels (fig. 6). In reality, they were large vacuoles (fig. 7) which replaced the nerve parenchyma,

the remnants of which plastered the walls (figs 5 and 6). In some instances the large vacuoles contained spirals and remnants of blood vessels (fig. 7), nerve fibers and broken up myelin or axons. One gained the impression that the nerve became fragmented by an immensely proliferated and infiltrated endoneural membrane which totally replaced some portions of the nerve. This appeared either as islands of transversely cut nerve fibers or as the previously described

Fig. 8.—Perineural space (*P.P.P.*) bordered by a folded arachnoid (*Ar*) and a villus-like (*V*) formation; at *E*, a thickened and vascular endoneural membrane.

vacuoles enclosing remnants of such islands (fig. 5) or as a hyperplastic and infiltrated endoneural membrane which took up the larger part of the nerve parenchyma (figs. 3 and 8). The entire nerve was surrounded by a perineural space which was bordered by the arachnoid (fig. 8). This appeared folded as if about to form a villus. The space was filled by a delicate network of trabeculae enclosing blood cells and an ill defined mass.

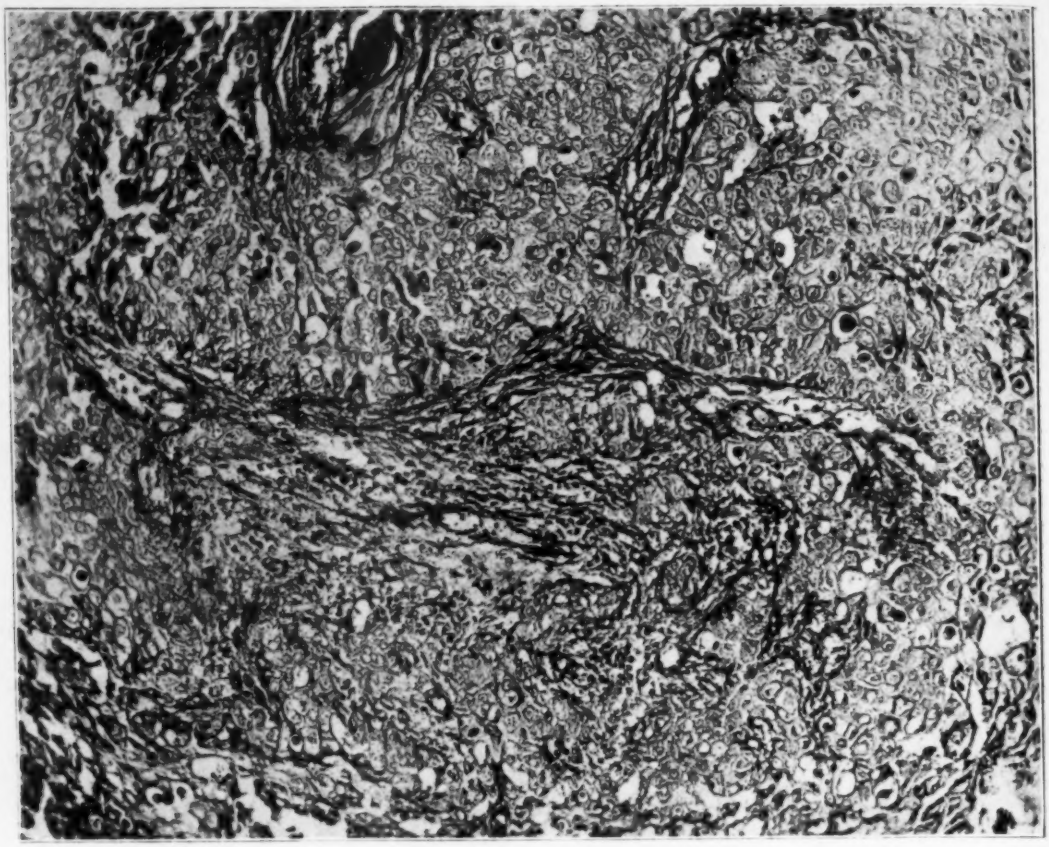

Fig. 9.—General aspect of the nerve described in the text. Same stain as in figure 3.

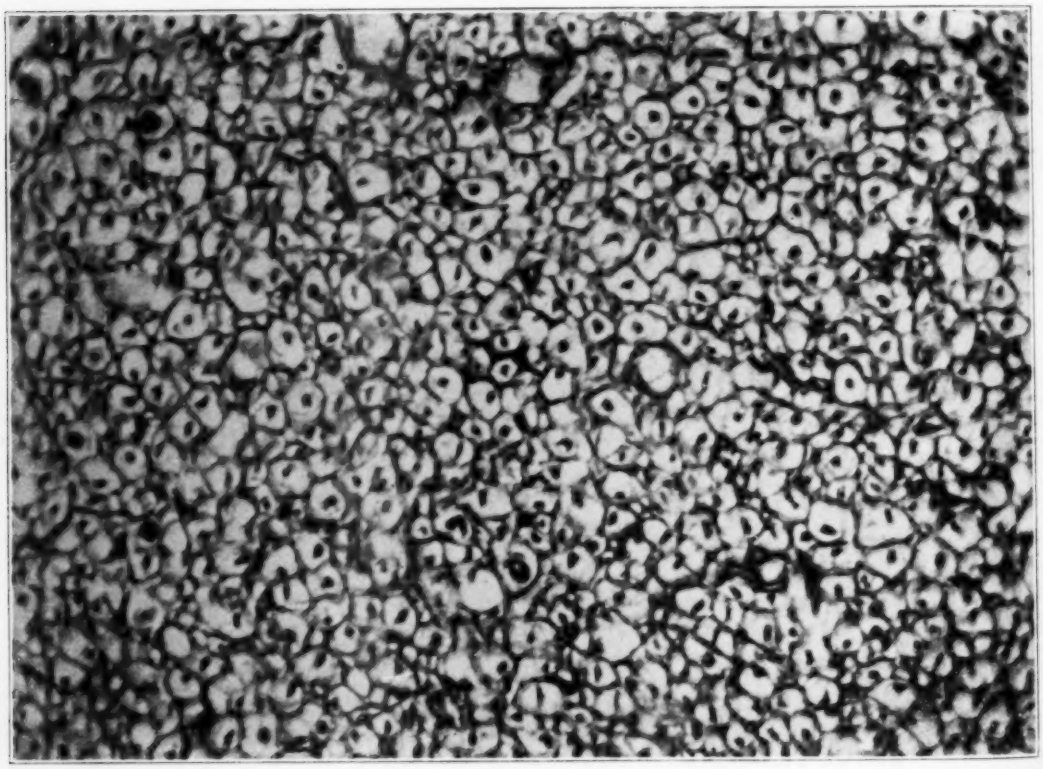

Fig. 10.—Normal nerve. Alzheimer-Mann stain.

The general aspect of the nerve was greatly changed. It differed greatly from a normal third nerve as shown in figure 10. In the latter the transverse outlines of the nerve fibers are clearly defined. The nuclei of the Schwann cells are in the background, and the nerve as well as the endoneurium is free from blood cells or blood vessels.

Examination of the brain itself showed no changes; the meninges, especially at the base, exhibited a mild infiltration with lymphocytes and plasma cells.

SUMMARY AND COMMENT

The changes in the nerve were degenerative, with generally a rather mild reaction on the part of the Schwann cells, but a severe reaction on that of their endoneurium and blood vessels. In other words, there was here a combination of intensive inflammatory and degenerative phenomena, a condition that may be defined as an interstitial neuritis, probably syphilitic. The vascular condition of the third nerve was evidently a partial manifestation of cardiovascular changes elsewhere in the body, in which a syphilitic aortitis was present with arteriosclerosis of the kidneys, heart and the large blood vessels of the brain. It is rather hard to tell what was the relationship between the two conditions—the nerve destruction and the interstitial phenomena. Nor is it possible to define with absolute certainty the nature of the changes in the artery. It was much like that seen in arteriosclerosis; yet it could also be caused by syphilis, for old syphilitic vascular changes may resemble those seen in arteriosclerosis (Jakob[1]). A syphilitic condition therefore cannot be denied here altogether.

One also might assume that some of the changes in the third nerve here described were due to pressure on it by the sclerosed posterior cerebral artery. However, in a pure nerve pressure lymphocytes and plasma cells are scarce; they are by no means so numerous as in this case. They were, for instance, absent in the opposite third nerve, which though compressed by a similar blood vessel showed no changes. In the literature, however, such an etiology—pressure on the nerve with consequent atrophy—has been occasionally mentioned. For instance, in Allbutt's[2] patient, a syphilitic subject, the third nerve was "simply thickened and condensed by increase in its connective tissue and its nervous fibers were consequently atrophied." While Allbutt saw nothing "distinctive" in that process, Baumgarten[3] considered such a condition

1. Jakob, A.: Normale und pathologische Anatomie und Histologie des Grosshirns, Vienna, Franz Deuticke, 1927, vol. 1, pp. 347 and 353; 1929, vol. 2, p. 578.

2. Allbutt, J., and Taylor: Case of Cerebral Disease in a Syphilitic Patient, St. George's Hosp. Rep. **3**:55, 1868.

3. Baumgarten, P.: Ueber gummöse Syphilis des Gehirns und Rückenmarks, namentlich der Gehirngefässe, und über das Verhältniss dieser Erkrankungen zu den entsprechenden tuberculösen Affectionen, Virchows Arch. f. path. Anat. **86**:179, 1881.

in his patient a gummatous neuritis. He stated that one does not deal here with "ordinary penetration of connective tissue, but with a gradual gummatous proliferation of the perineurium." This is infiltrated with "small" cells, the nerve parenchyma gradually becoming transformed into granulation tissue and the process progressively extending from the margin of the nerve to its interior.

Nothnagel's [4] patient presented a slowly developing bilateral paralysis of all the branches of the oculomotor nerve. Necropsy showed a bilateral dilatation of the posterior cerebral artery which produced pressure atrophy of both oculomotor nerves (the whole article is contained in six and one-half lines).

In one of Siemerling's cases,[5] both oculomotor nerves were degenerated; the left was transformed into a "nuclear" connective tissue. Numerous new-formed blood vessels were found; the nerve, on transverse sections, appeared shrunken; the nerve fibers were sparse; their myelin was changed, devoid of concentric layers and mostly without axons. In another case the nerve, near the exit from the peduncles, showed vascular proliferation, and many oculomotor nerve fibers were atrophied.

In Wilbrandt and Saenger's case [6] the third nerve showed minute hemorrhages; the blood vessels were so abundant that the condition resembled a "telangiectasis of the oculomotor nerve." The membranes of the nerve were thickened, so that one could not speak of gummatous infiltrations. The vascular phenomena, however, such as "hyperemia, hyperplasia," were pronounced and were, in the opinion of the authors, responsible for the degenerative state of the nerve.

In Papadaki's case [7] of paralysis of the oculomotor nerve, the ptosis was recurrent, but it finally remained stationary for twenty-one days until death. At necropsy the nerve was found enveloped by a band of meningitic connective tissue. In addition, a blood vessel with sclerosed walls "distorted" the nerve at the point of its emergence from the cerebral peduncle. It left two imprints which gave an image, probably fictitious, of a double strangulation. In one nerve many blood vessels were engorged, and numerous extravasations, old and recent, were present. Papadaki added that a nerve "fixed" can no longer escape the pressure exercised by large sclerosed blood vessels.

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4. Nothnagel: Anzeiger der K. K. Gesellsch. d. Aerzte in Wien, 1884, p. 92.

5. Siemerling, E.: Zur Syphilis des Centralnervensystems, Arch. f. Psychiat. **22**:191, 1891.

6. Wilbrandt, H., and Saenger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1900, vol. 1, p. 322.

7. Papadaki, A.: Paralysie du moteur oculaire commun chez un tabétique; examen anatomique, Rev. neurol. **12**:585, 1904.

In the cases quoted, the etiologic factors were much more evident and definite than in my case. As in Baumgarten's case, the etiology of the paralysis lay in the disease of the nerve itself; such causes as are given by the foregoing observers (basilar meningitis, strangulation of the nerve by bands of connective tissue, hemorrhages, lesions of the brain tissue proper) were absent in my case. The morbid process was in the nature of an acute inflammation, the cause of which it was not possible to determine. Syphilis, which may give such a histologic picture, should be considered in this case because of the presence of a syphilitic aortitis. Whether of syphilitic, arteriosclerotic or of any other etiology, the possibility of a primary nerve lesion should be borne in mind whenever the cause of a paralysis of the third nerve is obscure and the treatment unsuccessful. In my case treatment was unsuccessful because the nerve was replaced by connective tissue; it was thus hopelessly destroyed, and for this reason was incapable of regeneration.

CONCLUSIONS

1. Among the etiologic factors of a peripheral paralysis of the third nerve, a diseased condition of the nerve itself should be considered.

2. Such a morbid condition may be due to syphilis and may appear as vascular hyperemia, with distention and infiltration with lymphocytes, plasma cells and similar elements.

3. The marked vascular lesions cause an extensive destruction of the nerve fibers and their replacement by connective tissue.

4. The prognosis for recovery is decidedly bad, for destroyed nerve fibers, replaced by connective tissue, cannot regenerate.

5. The prognosis is also bad for life, for a vascular lesion of a cranial nerve is usually a partial manifestation of a general vascular disease, such as arteriosclerosis of the kidneys, heart and other vital organs.

THE TECHNIC OF ENCEPHALOGRAPHY*

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AND
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Encephalography as a diagnostic procedure is well recognized in most neurologic clinics. It has also been suggested and frequently used as a therapeutic measure. There have been two profound objections to such use. The first objection is the severe reaction that the patient experiences during and after the injection of air. This is frequently characterized by severe headache, nausea and vomiting, often projectile in character, marked diaphoresis and occasionally collapse. Second, it has been considered dangerous in patients who show much increase in intracranial pressure, especially in the presence of choked disks, tumors of the brain and particularly lesions of the posterior fossa.

Any procedure that will minimize this reaction is of value in the welfare of the patient as well as in facilitating the examination. It has been noted that to some extent the distress of the patient is in proportion to the amount of movement to which he is subjected. With this in mind, a chair was constructed in which the patient can be placed before the procedure and in which he can remain until he is returned to the ward.

The chair is so constructed that the patient can be placed in either an upright or a prone position which can be varied at will while he remains in the chair. It also has an adjustable head rest which will hold the head firmly during the injection of air and may be adjusted to any position during the procedure. This head rest may also be entirely removed during the making of the x-ray films. The chair is placed on a platform sufficiently large to counterbalance any change in weight caused by shifting the position of the patient. The platform is in turn mounted on large casters which simplify the movement of the patient from the operating room to the x-ray room and from the x-ray room

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* The chair and x-ray apparatus were obtained through the cooperation of Dr. Preston M. Hickey, of the X-Ray Department.

to the ward. The mechanics of the chair are illustrated in the drawings and in the pictures which show the various possible positions.

The puncture can be done with the patient prone or upright in the chair. In either case, he is seated for the injection of air. The preoperative medication and the technic for the introduction of air and the removal of fluid is more or less a matter of personal choice. Several good methods have been described.[1] Our preoperative medication varies considerably with the patient and with the condition that we expect to find. In cases of increased intracranial pressure, and par-

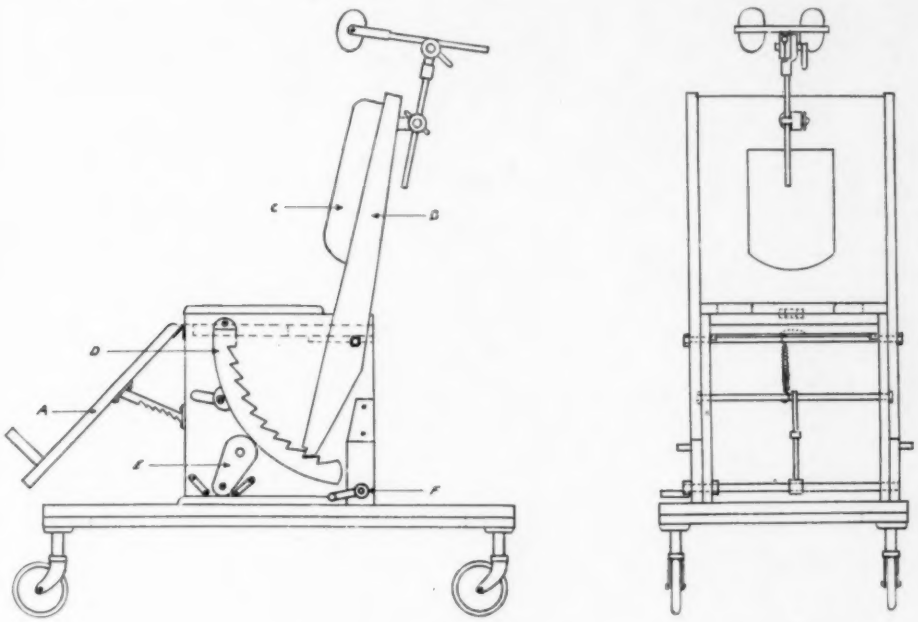

Fig. 1.—Diagram of the adjustable chair. *A* shows the adjustable leg support; *B*, adjustable back rest (by proper adjustment of *A* and *B* the patient can be made to sit upright or to lie prone); *C*, padding for the back; *D*, ratchet arm for holding the back; *E*, ratchet arm lock (ratchet arm and ratchet arm lock are on both sides of the chair), and *F*, foot pedal for releasing the ratchet arm.

ticularly in those in which tumor of the brain is suspected, we use either a solution of iso-amyl ethyl barbituric acid, given intravenously,

1. Schinz, H. R.: Zentralbl. f. Chir. **49**:1367 (Sept. 16) 1922. Bingel, A.: Deutsche med. Wchnschr. **47**:1492 (Dec. 8) 1921. Sicard; Gally and Haquenau: Bull. et mém. Soc. méd. d. hôp. de Paris **50**:1563 (Nov. 18) 1926. Kauffmann, H.: Beitr. z. klin. Chir. **136**:649, 1926.

the dose being proportional to the weight of the patient, or we use fairly large doses of chloral hydrate and sodium bromide. These drugs are also proportional to the weight of the patient and are given in physiologic solution of sodium chloride by rectum. In cases of epilepsy and in other types in which there is no increase of pressure,

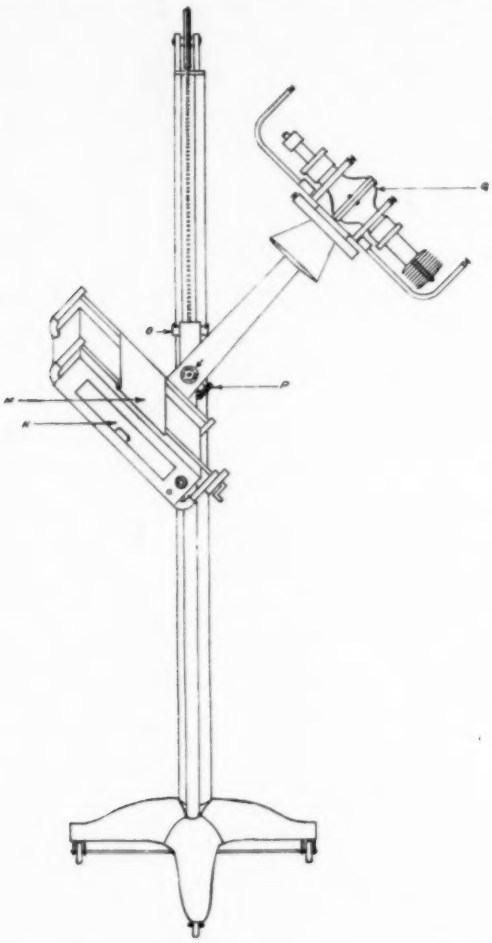

Fig. 2.—Diagram of the adjustable stand for x-ray tube and Bucky diaphragm. *M* shows the canvas band for immobilization of the head; *N*, plate holder; *O*, adjustment for raising and lowering the tube and diaphragm; *P*, adjustment for changing the angle of the arm which supports the x-ray tube and the Bucky diaphragm (the tube and Bucky diaphragm are always in constant relationship with each other), and *Q*, x-ray tube.

we use a combination of morphine or codeine and scopolamine. We use the two-needle method with a water manometer attached to the

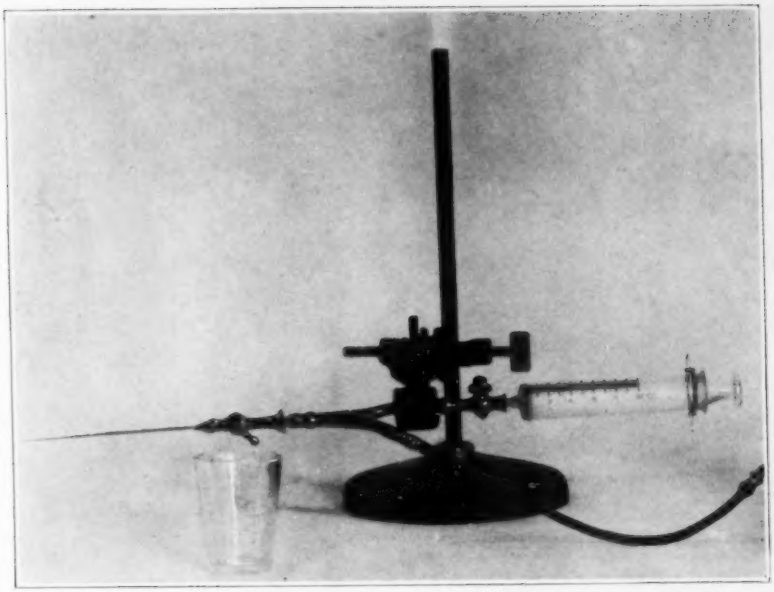

Fig. 3.—Showing connections of needle Y tube with syringe and tube to manometer.

Fig. 4.—Front view of chair.

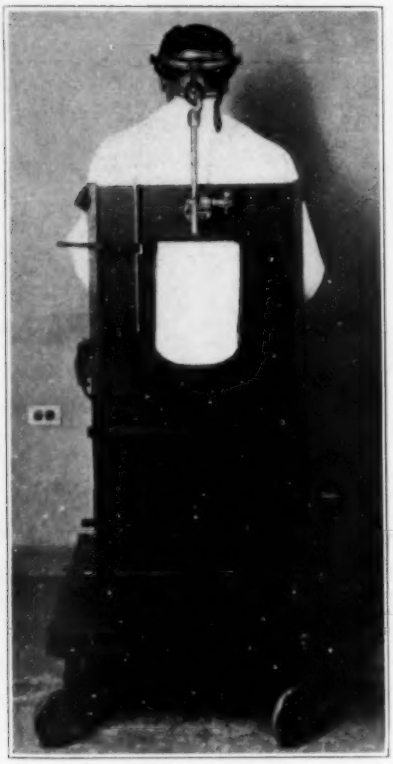

Fig. 5.—Back view of chair, showing head rest in position, manometer and holder.

Fig. 6.—Showing position of chair and tube stand for postero-anterior film.

Fig. 7.—Showing position of chair and tube stand for anteroposterior film.

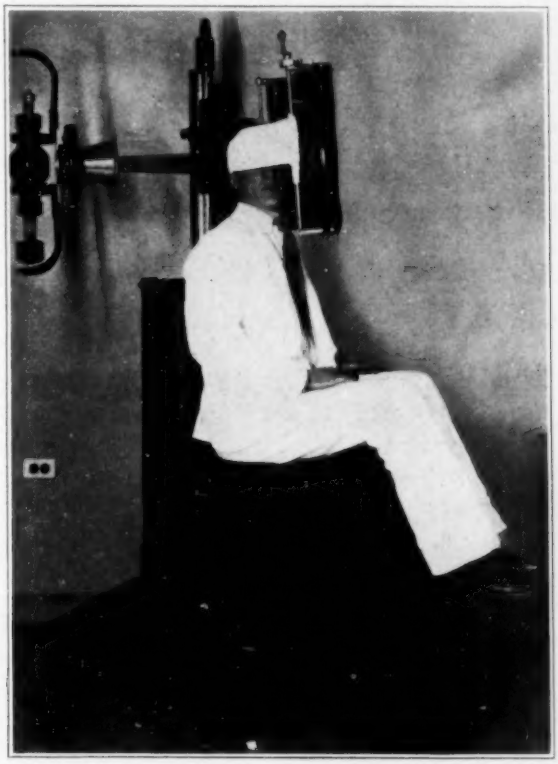

Fig. 8.—Showing position of chair and tube stand for lateral film.

upper needle or a single needle with a Y tube and the manometer attached to one side of the Y. Care in the rate of withdrawal of the fluid and of injection of the air is of prime importance. It is also important to move the patient's head forward and backward and from side to side near the end of the procedure to drain as much fluid as possible from the ventricles and subarachnoid spaces.

A second device which has been of great assistance is a special x-ray tube and Bucky diaphragm stand. This stand makes possible a constant x-ray technic which not only facilitates the mechanical part

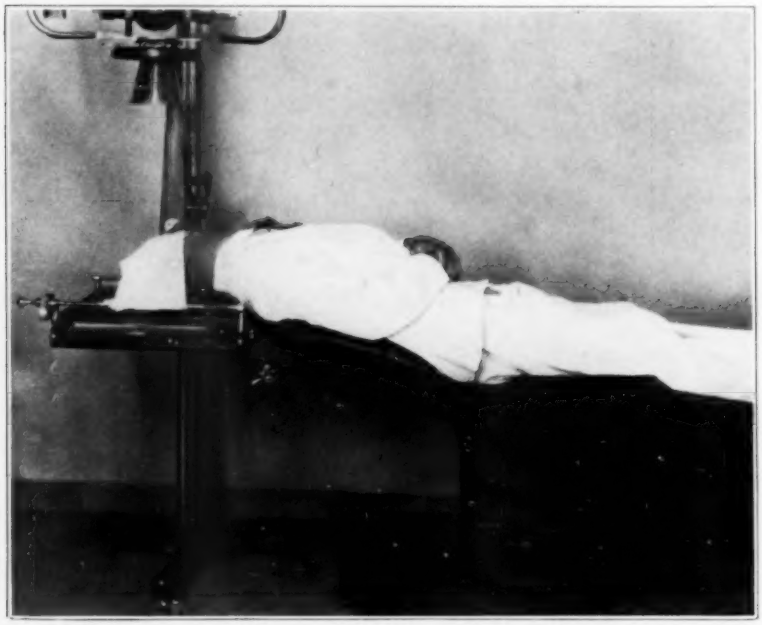

Fig. 9.—Showing position of chair and tube stand for lateral horizontal film to demonstrate inferior horn.

of the procedure, but also standardizes the films by making the relation of the patient's head, the x-ray tube and the film constant, thus making interpretation much easier.

The second objection to the use of encephalography is, in our opinion, becoming less important since the procedure may be carried out in cases of increased intracranial pressure. For example, a patient was admitted to the University Hospital complaining of severe headaches of three weeks' duration. Neurologic examination gave essentially negative results. The patient's general appearance was suggestive of

acromegaly. It was decided to make a ventriculogram in order to localize the tumor, but these studies were unsatisfactory. The pressure of the patient's spinal fluid in the prone position was 550 mm. of water, and there was a neuroretinitis with a choked disk of 2 diopters. An encephalogram was made under these conditions. One hundred cubic centimeters of fluid was removed and a similar amount of air was introduced. On the basis of the encephalogram, a diagnosis of right frontal tumor was made. The diagnosis was confirmed by an operation.

ABSTRACT OF DISCUSSION

Dr. Temple Fay, Philadelphia: I believe that Dr. Waggoner has made a real contribution to the technic of encephalography by devising a suitable chair for the process. Crude attempts have been undertaken elsewhere, but this is the most elaborate, and probably the most efficient, apparatus so far devised. The patient's reactions from encephalography are determined by several factors, and Dr. Waggoner has overcome one of the objectionable features in making the chair adaptable to various positions. We have found in preparing patients for the procedure that 10 grains (0.6 Gm.) of chloral and 20 (1.3 Gm.) of bromide given before and following the encephalogram greatly relieve the severe headache that invariably follows.

Another important factor in the suitability of the chair is the ease with which the patient's head may be adjusted. Dr. Stookey emphasized the necessary position of the neck in order that the air may enter properly, and it will be found that if the head is flexed on the chest during the period of air introduction, and the chin elevated during the drainage period, there is a much more rapid interchange between fluid and air.

The most important consideration in this procedure is the understanding of the contraindications. I believe that the procedure should never be begun with the patient in an upright position. Dr. Waggoner's chair offers the possibility of obtaining the initial pressure in a prone position, which should be done in cases in which there is high pressure, because if the patient is in a sitting position, withdrawal of the fluid is dangerous. One other contraindication is the use of the syringe to withdraw fluid. The creation of negative pressure is a dangerous factor, so that the replacement method, whether one uses a three-way stopcock or the two needle method, is immaterial if the indications or contraindications are carefully watched.

Dr. Percival Bailey, Chicago: I learned the tube-replacement method from Dr. Bleckwenn when in Madison, Wis., and I believe that he thinks that he originated it. I may be misquoting him.

Dr. Emanuel D. Friedman, New York City: We all must agree that Dr. Waggoner has introduced an important element into diagnostic procedure, because the standardization of technic will enable us to compare results. We have tried at the Mt. Sinai Hospital from the outset of our work to obtain a similar table, but unfortunately we were not so successful as Dr. Waggoner has been.

I was glad to hear him stress another important factor in the technic of encephalography, namely, the necessity of injecting the air slowly. Dr. Loeser, the house physician at Mt. Sinai Hospital, has developed a method of simultaneous replacement which makes use of only one needle; it has worked very satisfactorily.

I think, too, that the coincidental phenomena which develop during the procedure and immediately following it can be minimized to a great extent by sedatives and immobilization of the head.

DR. R. W. WAGGONER: In regard to Dr. Fay's question, the head rest makes it possible to change, very carefully, the position of the patient's head. Vomiting appears to be inversely proportional to the care exercised in the movement of the patient's head. Very few of our patients have had attacks of vomiting in the operating room; that occurs usually, if it occurs at all, when the head is shifted for the making of the roentgenogram.

The chair enables the operator to make the puncture with the patient in the prone position, then the patient can be placed upright for the injection of air. We have followed this procedure in cases of very high intracranial pressure. In one case the fluid spilled over the top of the manometer, which registers 750 mm. of water, and there were apparently no disastrous after-effects.

ARSENIC IN THE SPINAL FLUID

QUANTITATIVE ESTIMATION FOLLOWING THE INTRAVENOUS ADMINISTRATION OF TRYPARSAMIDE AND SILVER ARSPHENAMINE *

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The arsenicals are accorded a prominent place in the list of therapeutic agents employed in neurosyphilis, but opinion is less uniform concerning the relative merits of individual preparations than concerning the group of arsenicals as a whole. This is not strange when one envisages the variation in the clinical manifestations of neurosyphilis. In addition to the well known factors of age, race, sex and antecedent therapy, the clinical aspects of the disease vary more or less constantly according to the clientele of the institution concerned.

If the hospital or clinic is frequented by members of the higher economic and educational strata of society, the probabilities are that these patients will seek advice earlier than those who are prevented by economic reasons or by failure to appreciate the results of neglect. The period that intervenes between initial infection and neurologic examination will be shorter, resulting in earlier detection, and more prompt institution of treatment. The underlying pathologic process being less extensive and less chronic, more favorable therapeutic results should ensue. Intimate liaison between a department of dermatology or syphilology, where disease is encountered in its early stages, and a department of neurology will bring about similar results.

Public hospitals and clinics, on the other hand, especially those supported by the state or municipality, in which are found a large proportion of chronic invalids, encounter the disease in its more chronic and incapacitating forms. The failure of the patients to realize the seriousness of the situation and the necessity for persistent and protracted treatment render them less cooperative and impair the results that might otherwise be obtained.

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* From the Department of Neurology, Columbia University, the New York State Psychiatric Institute and the Department of Biochemistry, Columbia University.

* Read at the Fifty-Sixth Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 10, 1930.

The clinical outcome—cure, arrest or improvement—varies more or less directly with the factors mentioned. An accurate interpretation of therapeutic results in neurosyphilis is connected with many difficulties and requires that consideration be given to the patient's age, the duration of the infection and the amount and variety of previous treatment; that a careful evaluation be made of the neurologic or psychiatric symptoms; that a determination be made of the topographic location and pathologic type of the lesions, and that a proper interpretation be made of serologic reactions in the blood and spinal fluid.

Although clinical results should constitute the basis for final judgment concerning the value of any method of therapeusis, the contributions of the laboratory serve to enrich knowledge of the biochemistry of arsenical preparations. It was not so long ago that the opinion was generally held that the choroid plexus offered an impenetrable barrier to the passage of arsenic into the spinal fluid. That view is now known to be incorrect.

In 1921, two of us (L. H. C. and C. N. M.[1]) began an investigation concerning the penetration of arsenic to the spinal fluid following the intravenous administration of silver arsphenamine. Determinations of arsenic were made on 239 fluids from 151 patients at intervals of from two to ninety-six hours following the intravenous treatments. Arsenic was detected qualitatively in from 82 to 91 per cent of the fluids and in from 70 to 85 per cent of the cases. Quantitative measurement of arsenic was possible in from 74 to 83 per cent of fluids and in from 60 to 78 per cent of cases.

At the time of the publication of these results, data were not available to warrant a statement concerning the period that arsenic might remain in the spinal fluid following its intravenous administration. Subsequent investigations disclosed that arsenic, in an amount sufficient for quantitative determination, may be found in the spinal fluid as long as forty-three days after it has been administered intravenously. A summary of these results is given in table 1, the data covering the periods of from ninety-six hours to forty-three days having been hitherto unpublished.

From our results and from data that have been furnished by other investigators one can compare the penetrability of arsenic to the spinal fluid after the injection of silver arsphenamine, neoarsphenamine and arsphenamine ("old salvarsan"). As a result of the examination of the spinal fluids from twenty-seven patients who had received neoarsphen-

1. Cornwall, L. H., and Myers, C. N.: Am. J. Syph. **7**:287 (April); **7**:629 (Oct.) 1923.

amine, Fordyce [2] and his co-workers were able to detect arsenic in amounts sufficient for quantitative measurement in 77 per cent of fluids, but in less than one half of these did the quantity exceed 5 mg. per hundred grams, the maximum quantity reported being 83.6 mg. From an examination of 257 spinal fluids following the intravenous administration of arsphenamine, these same workers [3] reported sufficient arsenic for quantitative determination in 77.5 per cent of the fluids. In approximately two thirds of these fluids the amount of arsenic was less than 5 mg. per hundred grams.

After comparing the amounts of arsenic and the frequency of its occurrence in the spinal fluid following the intravenous use of these three preparations—silver arsphenamine, neoarsphenamine and arsphenamine

TABLE 1.—*The Quantities of Arsenic in the Spinal Fluid After the Intravenous Administration of Silver Arsphenamine*

Interval	Number of Fluids	Average Arsenic in Mg. per 100 Gm. of Dried Specimen	Maximum Amount of Arsenic in Mg. per 100 Gm. of Dried Specimen
2 hours	115	11.548	143.0
24 hours	45	7.185	155.0
48 hours	46	7.912	74.6
72 hours	33	12.589	192.0
96 hours	5	12.46	51.2
7 days	2	65.40	.....
11 days	1	27.70	.....
12 days	1	11.90	.....
26 days	1	1.42	.....
43 days	1	1.96	.....

—we believe that the evidence is conclusive that the silver preparation has the advantage of a higher index of penetration to the spinal fluid. This fact combined with its low degree of toxicity and its therapeutic efficacy makes it the preparation of choice in syphilis of the central nervous system.

For the purpose of the present investigation, five patients suffering from the dementia paralytica type of neurosyphilis were selected by one of us (H. A. B.) from the male service of the New York State Psychiatric Institute. These patients had not received any arsenical preparation for at least three months before the investigation was started.

2. Fordyce, J. A.; Rosen, I., and Myers, C. N.: Am. J. Syph. **7**:444 (July) 1923.

3. Fordyce, J. A.; Rosen, I., and Myers, C. N.: Am. J. Syph. **7**:478 (July) 1923.

Three grams of tryparsamide in 20 cc. of distilled water was administered intravenously, and lumbar punctures were performed at intervals of from 1 to 120 hours afterward. The results are displayed in tables 2 and 3. It will be noted that the arsenic content of the fluids

TABLE 2.—*The Amounts of Arsenic Recovered from Spinal Fluid After Intravenous Administration of Tryparsamide*

Serial Number	Time After Injection, Hours	Weight of Specimen		Total Solids, per Cent	Arsenic in Mg. per 100 Gm. Specimen	
		Moist	Dry		Moist	Dry
		Patient J. B.				
9036	1	9.7690	0.1040	1.06	0.01	0.96
9037	2	8.3665	0.0865	1.03	0.02	2.31
9038	3	8.6870	0.0870	1.00	0.01	1.15
9039	4	8.5085	0.0835	0.98	0.01	1.20
9040	5	8.1495	0.0895	1.09	Trace	Trace
9041	6	8.2160	0.0910	1.10	Trace	Trace
9042	12	7.8245	0.0815	1.04	0.05	4.92
9043	24	9.6465	0.1015	1.05	0.01	0.99
9044	48	6.7110	0.0710	1.05	0.01	1.48
9045	72	6.1430	0.0680	1.10	0.01	1.47
9046	96	5.9820	0.0620	1.03	0.01	0.81
9047	120	6.8000	0.7000	1.03	0.01	1.43
		Patient J. M.				
9081	1	7.4920	0.0770	1.02	0.08	7.80
9082	2	6.3830	0.0660	1.06	0.02	2.21
9083	3	6.2610	0.0710	1.13	0.01	1.41
9084	4	7.4970	0.0870	1.15	0.01	1.15
9085	6	8.3870	0.0870	1.04	0.006	0.57
9086	12	6.8710	0.0745	1.08	0.07	6.72
9087	24	8.5175	0.0875	1.03	0.04	4.00
9088	48	3.4635	0.0395	1.11	0.03	2.53
9089	96	7.8430	0.0780	0.79	0.01	1.28
9090	120	8.4920	0.0870	1.02	0.01	1.15
		Patient I. K.				
9059	1	4.3045	0.0455	1.04	0.02	2.22
9060	2	5.1655	0.0555	1.06	0.009	0.90
9061	3	5.5922	0.0572	1.02	0.07	7.00
9062	4	7.2650	0.0800	1.10	0.01	1.25
9063	6	9.8460	0.1160	1.17	0.02	1.72
9064	12	5.7112	0.0582	1.02	0.05	5.16
9065	24	6.2256	0.0715	1.14	0.02	2.09
9066	48	6.9647	0.0797	1.14	1.01	1.26
9067	72	7.3040	0.0790	1.08	0.02	1.90
9068	96	7.9930	0.0880	1.10	0.02	1.71
9069	120	7.1650	0.0750	1.04	0.05	5.33
		Patient V. B.				
9070	1	7.9547	0.0947	1.18	0.006	0.53
9071	2	7.3040	0.0920	1.26	0.04	3.26
9072	3	7.8240	0.0940	1.20	0.04	3.20
9073	4	9.6030	0.1230	1.27	0.05	4.07
9074	6	7.8975	0.0955	1.21	0.02	1.57
9075	12	7.5720	0.0870	1.15	0.05	4.59
9076	24	8.2860	0.1010	1.22	0.02	1.48
9077	48	6.9200	0.0850	1.22	0.05	4.12
9078	72	9.1420	0.1120	1.22	0.02	1.79
9079	96	4.4515	0.0565	1.26	0.02	1.77
9080	120	8.7093	0.1043	1.19	0.006	0.48
		Patient W. H.				
9048	1	5.9170	0.0600	1.01	0.02	1.66
9049	2	7.6355	0.0750	0.98	0.01	1.33
9050	3	5.9880	0.0595	0.99	Trace	Trace
9051	4	5.3875	0.0475	0.89	0.03	3.15
9052	6	6.2640	0.0610	0.97	0.01	1.64
9053	12	5.0082	0.0482	0.96	0.04	4.17
9054	24	3.9660	0.0410	1.03	0.04	3.68
9055	48	6.9800	0.0615	0.88	0.02	2.44
9056	72	8.1710	0.0790	0.96	0.02	2.53
9057	96	8.0120	0.0785	0.97	0.02	1.91
9058	120	8.0622	0.0782	0.97	0.02	2.56

was fairly constant, averaging between 1 and 3 mg., with the exception of the twelve-hour interval, when it was slightly in excess of 5 mg. The results indicate that the peak of the arsenic content of the spinal fluid occurred twelve hours after the tryparsamide was given. The maximum amount of arsenic was 7.8 mg., which occurred in patient J. M. at the one-hour interval.

The dose of silver arsphenamine administered to the patients who were used for the investigations summarized in table 1 was, in the vast majority of instances, 0.2 Gm., whereas the dose of tryparsamide was 3 Gm., or a dose fifteen times greater. The amount of arsenic recovered in the spinal fluid after the administration of silver arsphenamine was from three to eight times more than that recovered after tryparsamide.

TABLE 3.—*Average Amounts of Arsenic in Spinal Fluids Following the Intravenous Administration of Tryparsamide*

Interval After Injection in Hours	Number of Fluids	Average Arsenic in Mg. per 100 Gm. of Dried Specimen
1	5	2.636
2	5	2.002
3	5	2.552
4	5	2.164
6	5	1.11
12	5	5.112
24	5	2.448
48	5	2.366
72	4	1.922
96	5	1.496
120	5	2.19

CONCLUSIONS

1. Arsenic penetrates the spinal fluid in larger quantities after the intravenous administration of silver arsphenamine than it does after neoarsphenamine, arsphenamine or tryparsamide.

2. With a dosage of silver arsphenamine representing one fifteenth that of tryparsamide, from three to eight times as much arsenic can be recovered from the spinal fluid. In isolated instances the difference is much greater. The maximum amount of arsenic recoverable by us in any case after tryparsamide was 7.8 mg. per hundred grams; the maximum amount after silver arsphenamine was 192 mg. per hundred grams.

3. The favorable clinical results that have been reported in neurosyphilis as a result of treatment with tryparsamide must be ascribed to factors other than the actual quantity of arsenic that enters the spinal fluid.

ABSTRACT OF DISCUSSION

Dr. Bernard Sachs, New York: I have been much interested in this subject for a number of years. I am pleased to see that the authors have had greater success in proving the amount of arsenic in the cerebrospinal fluid (one hundred and twenty hours, if I am correct, after the last administration of the drug) than was possible a number of years ago. I also think it is fortunate that they have succeeded in proving that more arsenic can be recovered from the use of silver arsphenamine than from tryparsamide. I think that anything that will militate against the use of tryparsamide is a fortunate circumstance, particularly because of the wretched effect that this drug has on the optic nerve.

Dr. Lawrence S. Kubie, New York: Some years ago Voegtlin used a method of biologic assay of the amounts of various spirocheticidal agents that penetrated into the cerebrospinal fluid. He tested the spirocheticidal action of cerebrospinal fluid from treated cases. As I remember them the result of his observations was that sulpharsphenamine seemed to have the greatest power of spirocheticidal penetration into the cerebrospinal fluid. Have Dr. Cornwall and his associates made any comparison with silver arsphenamine?

Second, were the patients subjected to repeated lumbar puncture and did the data presented for different hours after injections represent repeated punctures on the same patients or a single puncture on different patients at different intervals? I ask that because I was interested at one time in the possibility of increasing the penetration of arsenicals into the cerebrospinal fluid by forced drainage. With the Koll and Youman method of determining arsphenamine itself, I found that the amount of arsphenamine which penetrated could not be appreciably increased by forced drainage. That puzzled me until I looked into the question of the physical state of the arsenic compound in the blood serum, and found out what had already been pretty thoroughly established, namely, that the arsenical forms some obscure colloidal combination with the protein of the blood stream for a short time, and then is distributed rapidly to certain organs such as the liver and bone-marrow. Therefore, the freeing of the arsenic for penetration into tissues which have been affected by disease must take place in some method which is different from any ordinary methods of physical diffusion.

Third, what method was used in determination of the arsenic? Lastly, as to the question of the rigidity of control; the number of cases in which the silver arsphenamine was used was not very great, and it is well known that when one is dealing with rather minute quantities of arsenic only a small amount remaining in needles and in glassware is enough to throw determinations out considerably. I do not mean to imply that this has happened. I would like a few more of the experimental data.

Dr. Charles R. Ball, St. Paul, Minn.: How do the clinical results of these two arsenical preparations compare?

Dr. Leon Hastings Cornwall: To answer the last question first, we did not intend in this presentation to touch on the clinical features. The clinical course of these patients was not followed by me. My own experiences with tryparsamide are, generally speaking, in accord with the published results of other workers in this field.

I have not done any personal work on sulpharsphenamine, but Dr. Myers has, and I will ask him to answer the question concerning the relative amounts of arsenic in sulpharsphenamine and silver arsphenamine.

There is just one remark that I would like to make about sulpharsphenamine. I feel that it has no place in the armamentarium for intravenous arsenic administration. The therapeutic dose is too near the lethal point. I have seen one or two sad results from the intravenous administration of this product, and I hope never to see any more. I never intend to use it again intravenously. I have in mind one case in which death ensued within twenty-four hours after the intravenous administration of less than 0.1 Gm., and I had not witnessed such a result since the early days of the use of arsphenamine. I think that this drug should be reserved for cases in which the intravenous administration of other arsenical preparations is impossible because of the condition of the veins and that it should be used intramuscularly only.

The serial punctures were done on the same patient. They were not isolated punctures on different patients.

I will ask Dr. Myers to answer the question concerning the method of arsenic determination and the control. Incidentally, Dr. Myers has been working on this problem for a period of ten years or more. His technicians are experienced at the work and his methods are very carefully supervised. He does the work in battery formation, sometimes several hundred at a time, and I feel that the technic is excellently controlled.

DR. MYERS: The late Dr. Fordyce and I carried out a great many experiments concerning the localization and fate of sulpharsphenamine in the viscera and its penetration into the central nervous system (Myers, C. N., and Fordyce, J. A.: *Am. J. Syph.* **9**: 18 [Jan.] 1925). These experiments were carried out so that they parallel the work on the various other arsenicals, such as the arsphenamines, and I might also add that they have been carried out on animals as well. As a result of our experience with the various types of arsenic preparation, it was found that the penetration of arsenic into the central nervous system and also into the spinal fluid following the use of sulpharsphenamine was comparatively small. These animal experiments were carefully controlled with the so-called normal. It is necessary to rule out extraneous sources of arsenic which may be ingested from the various types of foods which are fed to the animals.

As a result of these comparisons and the work which Dr. Cornwall and I have done with silver arsphenamine, it is safe to say that the penetration of arsenic into the central nervous system following the use of silver arsphenamine exceeds that of any drug which we now have at our disposal. Our investigations have shown that there is a difference in the penetration depending on the type of neurosyphilis with which we are dealing. This may be found in some of our reprints dealing with this subject. Our investigations show that arsenic may be detected in from 80 to 100 per cent of the fluids examined when we are concerned with lesions that are found in the mesodermal structures, whereas there is only from 60 to 90 per cent when the ectodermal tissues are involved. I recall that in one of our investigations arsenic was found in the spinal fluid at the end of two hours in amounts as high as 143 micromilligrams per one hundred grams of dried specimen. The methods which we have employed are accurate to about 1:1,000,000, so I think we are within the limits that could be desired for an analytical technic of this kind. The needles which were used in these experiments

were either new needles or those which were washed with physiologic solution of sodium chloride, which had previously been tested for arsenic, so we feel contamination through the needles was entirely eliminated.

Silver arsphenamine is particularly satisfactory on account of its high penetrability in the central nervous system, its convenience and also its lack of systemic effect.

MANIC-DEPRESSIVE PSYCHOSIS

THE RELATION OF HEREDITARY FACTORS TO THE CLINICAL COURSE *

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Although there are numerous reports on the hereditary factors in manic-depressive psychosis, on the age of the patient at onset, the length of the attacks and the length of the intervals, the reports dealing with possible correlations between the hereditary factors and the other factors mentioned are extremely rare. In a disorder, the hereditary factors of which are so prominent and so varied, it is only natural to ask whether a certain type of heredity is related to a certain clinical course of disease. In other words, can one predicate the occurrence of a severe clinical course on the presence of severe familial neuropathic factors, or is the course of the disease unrelated to these? A search of the literature revealed but few replies to these questions. In textbooks, such as those by Ziehen, Pilcz, Stransky, Strohmayer, Kraepelin, Bleuler and Lange, no possible correlation between these factors was mentioned. A thorough search of the periodic literature disclosed but one article on this subject, by Fitschen.[1] She studied 120 institutional cases and found that in patients showing severe hereditary tainting, the age of onset was earlier than in those in whom the hereditary tainting was not severe. Fitschen considered those patients severely tainted in whom a neuropathic taint was found in both parents, in two or more siblings or in parents and other members of the family. Since reports dealing with the aforementioned correlation are so rare, it seemed worth while to make a further study of this point.

The material studied here consists of the records of manic-depressive psychosis from the private practice of Dr. Hugh T. Patrick. In two previous communications [2] based on a study of this series, I reported the age incidence of first attacks, the length of the attacks and the length of the intervals. These values differed from similar

* Submitted for publication, March 26, 1930.

* From the Department of Nervous and Mental Diseases, Northwestern University Medical School.

1. Fitschen, E.: Die Beziehung der Heredität zum periodischen Irresein, Monatschr. f. Psychiat. u. Neurol. **7**:127, 1900.

2. Paskind, H. A.: Manic-Depressive Psychosis as Seen in Private Practice: Sex Distribution and Age Incidence of First Attacks, Arch. Neurol. & Psychiat. **23**:152 (Jan.) 1930; Manic-Depressive Psychosis as Seen in Private Practice: Length of Attack and Interval, ibid. **23**:789 (April) 1930.

ones in the literature, and these differences were explained by the fact that in this series are found large numbers of mild cases not requiring institutionalization, while the literature is based almost entirely on the observation of classic institutional cases, a variety that runs a much more malignant course than the cases dealt with here. In this series, the median value for the age of onset was 31 years; the median for the length of the attacks was four months; the median for the length of the intervals was seven years. The hereditary factors were noted in 485 cases; a study of these factors was made the subject of a separate report.[3] The present study is concerned with the question of whether or not different types of hereditary tainting are correlated with differences in the median values for the age of onset, for the length of the attacks and for the length of the interval.

Results of Reports on Six Hundred and Thirty-Three Patients Treated for Manic-Depressive Psychosis

	Number of Cases	Median Age of Onset, Years	Median Length of Attacks, Months	Median Length of Interval, Years
Entire series	633	31	4	7
Family history entirely free from taint	83	33	4	7
All cases with a neuropathic taint	402	30	4	7
Both parents with a neuropathic taint	69	22	5	4
Psychosis in a parent	113	29	4	5
Migraine in a parent	65	29	3	3
Nervousness in a parent	100	30	4	7
Neuropathic taint in a parent and siblings	164	30	4	6
Psychosis in a parent and in one or more siblings	24	30	4	6
Neuropathic taint in parent and grandparent on the same side	74	26	4	7

For purposes of tabulation, I divided the cases into nine groups, each group corresponding to a definite type of hereditary neuropathic tainting. I then determined in each group the median value for the age of onset, the length of the attacks and the length of the intervals. The results are shown in the accompanying table.

It appears that the most benign course of the disease is found in cases in which the family history is entirely free from taint. Here the onset tends to be comparatively late (median value, 33 years); the median for the length of the attack is four months; the median value for the length of the intervals is seven years. In instances in which both parents have a neuropathic taint, the disease runs a much more severe course. Here the onset is much earlier (median, 22 years); the attacks are longer (median, five months), and the intervals shorter

3. Paskind, H. A.: A Comparison of the Hereditary Factors in Institutional and Extramural Cases of Manic-Depressive Psychosis, Arch. Neurol. & Psychiat. **24**:747 (Oct.) 1930.

(median, four years). Cases in which migraine was detected in a parent had comparatively severe courses; the intervals were very short (median, three years), and the median value for the age of onset was two years below the median value for the entire group, and four years below the median value for cases in which no familial neuropathic taint was detected. Cases in which a psychosis was found in a parent were characterized by relatively short intervals (median, five years). Instances in which nervousness was found in a parent and cases in which a neuropathy was found in parents and siblings had median values corresponding well with that for the entire group. Cases in which a neuropathy existed in a grandparent in addition to a parent were characterized by a relatively early onset (median, 26 years).

CONCLUSIONS

Four hundred and eighty-five cases of extramural manic-depressive psychosis were studied to determine whether or not any correlation existed between familial neuropathic factors and the age of onset, the length of the attacks and the length of the intervals. A rather definite correlation was found.

Clinical Notes

OPHTHALMOPLEGIA INTERNUCLEARIS AND OTHER SUPRANUCLEAR PARALYSES OF THE EYE MOVEMENTS*

WILLIAM F. MONCREIFF, M.D., CHICAGO

The varieties of ophthalmoplegia that comprise the subject for consideration in this paper are produced by supranuclear lesions which impair lateral or side to side movements, vertical or up and down movements, or certain combinations of both. All of these types of movements are mediated by supranuclear mechanisms, lesions of which are characterized by symmetrical impairment of directional movements, or by dissociated paralyses, rather than by impaired function of a single muscle, or of a monocular group of muscles innervated by a single nerve or from a single nucleus.

PARALYSES OF LATERAL MOVEMENTS

Cortical or subcortical lesions of the second frontal gyrus may cause temporary paralysis of voluntary lateral eye movements, without affecting the reflex and automatic movements. The reflex conjugate movements in response to visual, auditory or equilibratory stimuli are impaired or destroyed by lesions of the corresponding centers or pathways, without affecting the voluntary movements, assuming that the frontal centers are intact. Both voluntary and reflex types of movements are affected by supranuclear lesions in the pons. Many authors assume the existence of a supranuclear pontile center for lateral movements, and consider that fibers from this center pass to the nuclei of the internal and external recti. According to Lutz,[1] this center is accepted by Bing,[2] Wilbrand and Saenger,[3] Holmes,[4] Cantonnet,[5] Grasset,[6] Julin,[7] Raymond[8] and many others. Lutz, however, stated that no one has so far been able to find such a center, and

* Submitted for publication, July 30, 1930.

* From the Department of Ophthalmology, Rush Medical College.

* Read at a Meeting of the Chicago Neurological Society, April 17, 1930.

1. Lutz, A.: Ueber die Bahnen der Blickwendung und deren Dissoziierung, Klin. Monatsbl. f. Augenh. **70**:213 (Jan.-Feb.) 1923.

2. Bing, Robert: Gehirn und Auge, Munich, J. F. Bergmann, 1923.

3. Wilbrand and Saenger: Die Neurologie des Auges, Munich, J. F. Bergmann, 1922, vol. 8.

4. Holmes, Gordon: Palsies of the Conjugate Ocular Movements, Brit. J. Ophth. **5**:241 (June) 1921.

5. Cantonnet, A., and Lapersonne, F.: Soc. de neurol., 1907.

6. Grasset, J.: Leçons cliniques, Montpellier, C. Coulet, 1898, p. 494.

7. Julin, G.: Les paralysies des oculogyres, Paris, 1909, p. 52.

8. Raymond, Cestan: Rev. neurol. **11**:644, 1903.

questioned whether there is any necessity for accepting such a center, a view that is shared by Dejerine[9] and Spiller.[10]

Different varieties of paralysis of lateral movements due to pontile lesions have been variously enumerated and described by Fuchs,[11] Duane,[12] Wilbrand and Saenger,[3] Spiller,[13] Lutz[1] and others. The existence of the following varieties seems to be definitely established:

1. Paralysis of lateral conjugate movement with retention of convergence. This form is due to a lesion of the posterior longitudinal bundle, involving the descending fibers to the abducens nucleus and the ascending fibers for conjugate lateral movement to the internus cells of the nucleus oculomotorius. The fibers for convergence innervation to the internus nucleus lie at a higher level and are spared. A unilateral lesion of this kind interferes with movement to the same side (*A* in accompanying figure). Loss of movement to both sides is produced by large or bilateral lesions, such as a lesion spreading from one side of the pons to the other, or a tumor extending from the midline to both sides.

2. Paralysis of lateral conjugate movement combined with paralysis of convergence function of the heterolateral eye. This form is due to a lesion involving the posterior longitudinal bundle and also extending upward to involve the supranuclear pathway for convergence. In these cases there is usually a large lesion which distorts the whole pons, with loss of lateral movement to both sides, as well as convergence (Freeman[14]).

3. Ophthalmoplegia internuclearis of l'Hermitte.[15] This consists, according to Lutz,[1] of the following two varieties: (*a*) Ophthalmoplegia internuclearis anterior, in which the externus on the side of the lesion functions normally, and the internus of the contralateral eye functions normally in convergence but is paralyzed for lateral conjugate movement (toward the lesion). In this type there is a lesion of the posterior longitudinal bundle which affects only the ascending fibers to the internus nucleus for conjugate lateral movement (*B* in accompanying figure). This form of paralysis may be bilateral (Wilbrand and Saenger[3]). (*b*) Ophthalmoplegia internuclearis posterior, in which both interni function normally in convergence and in lateral conjugate movement, but the externus on the side of the lesion is paralyzed for conjugate movement. The lesion is one affecting the descending fibers in the posterior longitudinal bundle to the abducens nucleus (*C* in accompanying figure).

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9. Dejerine, J. J.: Sémiologie des affections du système nerveux, Paris, Masson & Cie, 1914.

10. Spiller, W. G.: Ophthalmoplegia Internuclearis Anterior: A Case with Necropsy, Brain **47**:345 (Aug.) 1924.

11. Fuchs, E.: Textbook of Ophthalmology, Philadelphia, J. B. Lippincott Company, translated by A. Duane.

12. Duane, A.: The Associated Movements of the Eyes: Their Nerve Centers, Conducting Paths, Production, Varieties and Derangements, Am. J. Ophth. **7**:16 (Jan.) 1924.

13. Spiller, W. G.: Importance in Clinical Diagnosis of Paralysis of Associated Movements of Eyeballs, Especially Upward and Downward, J. Nerv. & Ment. Dis. **32**:417 and 497, 1905.

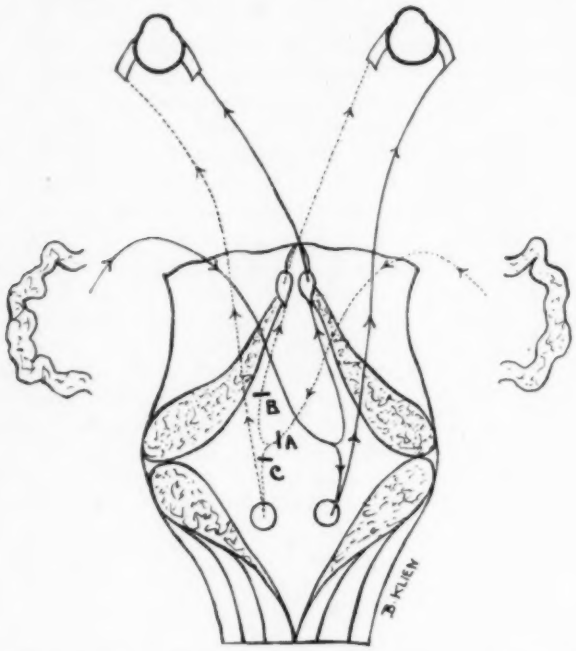
14. Freeman, Walter: Paralysis of Associated Lateral Movements of the Eyes: A Symptom of Intrapontile Lesion, Arch. Neurol. & Psychiat. **7**:454 (April) 1922.

15. L'Hermitte, cited by Spiller (footnote 13).

Ophthalmoplegia internuclearis anterior would appear to be a rather unusual form of paralysis of lateral movement, as Spiller,[10] who in 1924 reported a case with necropsy, was able to find only eleven cases reported up to that time. I have recently observed this condition in a patient admitted to the service of Dr. Peter Bassoe at the Presbyterian Hospital, with a diagnosis of encephalitis disseminata. Dr. Klien and Dr. Bassoe have given me permission to present this case.

REPORT OF CASE

History.—F. G., a boy, aged 15, was seen in the eye clinic of the Central Free Dispensary on Feb. 25, 1930, complaining of diplopia on looking to the left, and vertigo and unsteadiness in walking, of two weeks' duration. The same evening



A, lesion producing paralysis of conjugate movement to the left; *B*, lesion producing ophthalmoplegia internuclearis anterior; *C*, lesion producing ophthalmoplegia internuclearis posterior.

he was admitted to the service of Dr. Peter Bassoe with a tentative diagnosis of disseminated encephalitis. He vomited twice before entering the hospital and two or three times afterward. For two weeks prior to the onset of this attack, he had noticed peculiar sensations in the arms and face, and especially burning sensations on the left side of the face.

The past history was of interest, in that three similar attacks had occurred previously, each of which was characterized by diplopia, staggering gait and weakness and incoordination of the hands and arms, especially the right. The first and most severe attack, in which there was vomiting and also fever, was of about three weeks' duration, in May, 1929. The second attack, in August, was less severe than the first. During the third attack, which was in November, there

was diplopia on looking to the right. After recovery from this attack, a tonsillectomy was done. Early in childhood the patient had had measles, pertussis, pneumonia and influenza. The family history was without special significance.

Examination of the Eyes.—There was an unusual type of impairment of the movements; viz., on looking to the left, the right eye could not move beyond the midline, and the left eye exhibited coarse jerky nystagmic movements. Convergence movements and all other associated movements were normal. There was a spontaneous rotary nystagmus, which was slight with the eyes in the primary position but was markedly accentuated with the eyes in positions of extreme rotation to the right or left, and to a lesser extent upward or downward. The visual acuity of each eye was normal; there was no abnormality of the pupils or pupillary reflexes or of the fundi.

Physical, Neurologic and Laboratory Examinations.—The patient was rather undernourished and physically underdeveloped. There were no significant observations in the circulatory and respiratory systems. There was a slight leftsided facial paresis; also weakness and ataxia of the right arm, with intention tremor. The gait was staggering, both feet dragging in walking, and the patient swayed from side to side on attempting to stand erect whether the eyes were open or closed. The Babinski and Chaddock signs were positive on both sides; the Oppenheim and Gordon signs were negative. The abdominal reflexes were weak on the right side and absent on the left side. On lumbar puncture, the spinal fluid was clear and under normal pressure; the cell count was 2, and there was a trace of albumin by the Nonne and Ross-Jones tests; the serologic reactions were normal. The blood, urine and stools were normal.

Course.—There was practically no fever except for a temperature of 99.4 F. on the second day in the hospital. The pulse and respirations were normal. Lateral movements of the right eye to the left improved daily, and on the fifth day in the hospital were almost normal, while the nystagmus of the left eye in looking to the left was less marked. After ten days, there was no defect of rotation of either eye in any direction and no diplopia in any part of the binocular field, but there was slight nystagmus on looking strongly to the right, and more marked nystagmus on looking strongly to the left, the nystagmoid excursions of the left eye being greater than those of the right. It was noted that prior to spinal puncture on the second day in the hospital, the patient was very drowsy and also had vomited several times. After the puncture, vomiting ceased and the patient became more alert mentally. Sodium salicylate was given intravenously in doses of from 7 to 10 grains (0.46 to 0.65 Gm.) from February 27 to March 29, at first daily and after four days at less frequent intervals, for a total of sixteen doses. From April 1 to 4, inclusive, sodium cacodylate was given intravenously in doses of from 5 to 7½ grains (0.32 to 0.49 Gm.) daily.

On April 2, the labyrinthine functions were examined by the rotation tests and found to be normal; these tests were not made earlier for fear of exciting a recurrence of the vomiting. There were some variations in the intensity of the spontaneous nystagmus and also in the degree of weakness of the extremities at different times during the course. The left-sided facial paresis was more marked on April 12 than at any earlier time.

Comment.—While this case is presented as an example of ophthalmoplegia internuclearis anterior, there was evidently also some partial impairment of function of the supranuclear fibers to the abducens, since in looking to the left there was coarse nystagmoid movement of the left eye.

The presence or absence of binocular diplopia is not of itself a symptom which differentiates with certainty between supranuclear and nuclear or peripheral forms of paralysis. I may say, however, that cortical and subcortical lesions tend to produce symmetrical paralyses, hence without diplopia.

PARALYSES OF CONJUGATE VERTICAL MOVEMENTS

Paralyses of the conjugate vertical movements are of the following varieties: 1. Paralysis of upward movement only. This is the most frequent variety. 2. Paralysis of upward and downward movement combined. This is second in frequency. 3. Paralysis of upward or of both upward and downward movement, combined with some form of paralysis of lateral movement. 4. Paralysis of downward movement only. This variety is least frequent of all.

Paralyses of the vertical movements are much less frequent than those of lateral movement. Spiller,[13] in 1905, reported nine cases of the former variety, with thirty-eight additional cases collected from the literature.

There is no definite agreement as to the location of lesions causing paralyses of vertical movements. Spiller[13] attributed these palsies to lesions near the oculomotor nucleus, such as a tumor in the ventral part of the pons, causing pressure on the posterior part of this nucleus, where he assumed that the nuclei of the superior rectus and inferior oblique are situated. A more recently stated view is that of Holmes,[4] who denied that these paralyses can be satisfactorily accounted for on the basis of nuclear lesions, and concluded that they are due to lesions of supranuclear mechanisms. The location of these supranuclear mechanisms or centers is not definitely known, and is variously located by different authors as in the tegmentum, or the central gray matter around the aqueduct of Sylvius or, by others, in the anterior corpora quadrigemina. Holmes inclined to the latter view. Wilbrand and Saenger[3] stated, however, that it is doubtful whether a lesion of the corpora quadrigemina which does not compress the adjacent structures can cause paralysis of vertical movements. They emphasized that paralyses which are very regular and equal on the two sides are due to supranuclear lesions, while those which lack this symmetry have a nuclear component. Collier,[16] in 1927, reiterated this distinction, and stated further that it seems probable that the descending paths from the cortex for upward movement, downward movement and convergence cross in the posterior commissure, while those for lateral conjugate movement are situated more laterally and cross lower down. Wilbrand and Saenger[3] reported that in patients with paralyses of vertical movements of the eyes the following lesions have in various cases been found: tumor of the pineal body, tubercle of the thalamus, tumors on the floor of the aqueduct of Sylvius, destruction of the surface of the corpora quadrigemina by carcinoma and glioma of the corpora quadrigemina.

Holmes[4] observed that tumors and other lesions extending from before backward into the midbrain disturb first the upward movements of the eyes, then downward movements and finally convergence, from which it would appear that the center (or pathways) for elevation lies anterior to that for depression. He also stated that loss of elevation is usually combined with disturbance of pupillary reactions to light, and loss of downward movement frequently with loss of convergence and accommodation. He referred to the dissociated palsies of the vertical movements, the most common form of which is loss of the voluntary

16. Collier, J.: Nuclear Ophthalmoplegia, with Special Reference to Retraction of Lids and Ptosis, and to Lesions of Posterior Commissure, Brain **50**:488 (Oct.) 1927.

movements, with reflex movement intact. In these dissociated palsies, he assumed an interruption, in the area adjacent to the supranuclear center, of the cortico-bulbar fibers that terminate within it.

SUMMARY AND CONCLUSIONS

To summarize, dissociated paralyses, as of voluntary movements, or of reflex conjugate movements in response to visual, auditory or equilibratory stimuli, are caused by lesions of the corresponding centers. Supranuclear lesions in the pons impair all varieties of reflex movement and voluntary movement, together. The normal lateral movements and their disturbances can be explained without assuming a supranuclear center.

The conjugate paralyses of lateral movement may be grouped in three divisions: (1) paralysis of lateral movement with retention of convergence; (2) paralysis of lateral movement combined with paralysis of convergence; (3) ophthalmoplegia internuclearis, (*a*) anterior and (*b*) posterior. The clinical significance of these paralyses with regard to surgical indications has been well stated by Freeman,[14] who emphasized that clearcut paralysis of conjugate lateral movement with retention of convergence is characteristic of a lesion within the pons, and has not been produced by a cerebellopontile angle (or acoustic) tumor.

Paralyses of upward and of downward movement are much less frequent than those of lateral movement, and may occur separately, combined with each other or in combination with various forms of paralysis of lateral movement. The location of supranuclear lesions causing paralysis of vertical movements is not definitely known.

It seems most probable that these supranuclear mechanisms are located in the immediate vicinity of, but not in, the corpora quadrigemina. Paralyses of vertical movements have been reported as occurring in association with lesions of the pineal body, thalamus, tegmentum of the pons, the floor of the aqueduct of Sylvius, the posterior commissure and tumors of the corpora quadrigemina which compressed the adjacent structures.

55 East Washington Street.

PRIAPISM AS AN EARLY SYMPTOM IN MULTIPLE SCLEROSIS *

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ROCKFORD, ILL.

Multiple sclerosis is a common disease,[1] and hence the importance of recognizing its early symptoms is worth emphasizing. In several series of cases [1] the literature shows that the average symptom-bearing duration, from the onset to actual recognition of the disease, varied from three weeks to fourteen years, with an average

* Submitted for publication, July 19, 1930.

* Read at a Meeting of the Winnebago County Medical Society, May 23, 1930.

1. Wechsler, I. S.: Statistics of Multiple Sclerosis, Arch. Neurol. & Psychiat. **8**:59 (July) 1922; Report of Association for Research in Nervous and Mental Disease, New York, Paul B. Hoeber, Inc., 1922, vol. 2, p. 33.

of four years. This means that these cases might have been recognized four years earlier with a proper appreciation of early symptoms. In the interest of knowledge concerning these early manifestations, we desire to draw attention to a bizarre early symptom of multiple sclerosis, namely, priapism of a chronic nature lasting for a period of years. Wechsler [2] mentioned that this occurs, but gave no details. No other author of a textbook has mentioned the symptom, but the literature supplies two cases to which we now draw attention and then report an additional case.

CASES FROM THE LITERATURE

CASE 1.—A boy, aged 7, developed difficulty in standing and walking following an attack of measles. A little later he began to use his hands with a certain awkwardness and complained of headaches and poor vision. This early condition improved at times, but each time for a short period only. When first examined, the pupils were normal, but there was optic atrophy on both sides. The upper extremities showed ataxia, but were normal otherwise. The lower extremities revealed a flaccid paralysis, but there was no edema or vasomotor disturbance. The skin was somewhat scaly and rough. The extremities could be moved readily by passive motion. The knee jerks were absent; the achilles jerks were increased; an abductor reflex was present; the cremasteric reflex was absent; a Babinski sign was present. The skin of the abdomen, from the ribs to the umbilicus, showed a mild hypesthesia and hypalgesia. The skin below the navel showed no sensation to pain or touch; pin pricks produced only a reflex jerking of the legs. The sense of temperature was apparently normal. Muscular sense could not be tested on account of lack of cooperation. Priapism was almost constant, and there was incontinence of urine (urinalysis gave negative results).

The symptoms and signs of the disease showed a steady progress, which was interrupted by short remissions. The symptom-complex changed frequently, but eventually there was spastic paralysis of both lower limbs. As to the upper limbs, paralysis and ataxia alternated. There was marked disturbance of sensibility, with bladder and rectal disturbance. Priapism continued and optic neuritis developed; a marked speech defect came on and the hearing became impaired; the muscles of the eye, as well as the facial muscles, showed paralysis; finally, there came progressive dementia, which continued over a period of eight months until death. Priapism was continuous from the start.

At autopsy, this pathologically was a case of multiple sclerosis. In the spinal cord, medulla, midbrain, cerebellum and cerebrum were numerous areas of broken-down nerve tissue; many axis cylinders were uncovered in places; the ganglion cells showed a secondary mild degeneration; there was an overgrowth of glia cells.

CASE 2.—A man, a native of Chicago, aged 67, gave a history of priapism for eighteen years.[3] He was engaged in ice packing when the symptom first was noticed. He would awake three or four times during the night, but had no erections during the day. He would stay awake at night in order to avoid the painful occurrence. Sexual intercourse was not painful, nor did it have any apparent effect on the painful erections. Pinching the inner thigh gave a good deal of relief, as did cold compresses applied to the perineum and back of the scrotum. The patient stated that the condition was getting worse, the erections

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2. Wechsler, I. S.: A Textbook of Clinical Neurology, Philadelphia, W. B. Saunders Company, 1928, p. 135.

3. Kretschmer, H. L.: Demonstration Before the Chicago Neurological Society of a Case, J. Nerv. & Ment. Dis. **29**:427 (Jan.) 1909.

coming on as he went to bed and becoming more and more painful. Two weeks before admission, while turning on the gas light during work, he noticed that he was reaching for the gas jet with a broom handle. Since then he had had the feeling in the left hand as though he wanted to catch hold of something. If he concentrated his mind on the hand it would remain still, but when he walked —as he did with the aid of a crutch in the left hand—the right hand would continuously go through the motion of grasping something until he thought of it.

A physical examination gave negative results.

Neurologic examination revealed a slight nystagmus. The left achilles reflex was absent; there was no ankle clonus; a Babinski sign was present on the right; the right knee jerk was ++; the cremasteric reflexes were present; the right abdominal reflex was reduced. The special senses were normal except that the patient had difficulty in recognizing the difference between heat and cold on the skin on the penis.

The patient failed to respond to treatment, and left the Alexian Brothers Hospital in a discouraged frame of mind and was not seen thereafter.

REPORT OF PERSONAL CASE

History.—A married man, in the early thirties, who followed an occupation that called for close mental and physical application and strain, had been more nervous and upset than usual for some time. In addition, he had been undergoing unusual strain in a difficult piece of automobile driving for some days. He was of a nervous type, conscientious and ambitious, and always threw himself wholeheartedly into any mental or physical problem at hand.

He was taken to a hospital by a physician who found him in great pain from priapism that had been present in some degree for several days, but had grown worse so that he was unable to be about. Moreover, he was delirious and needed constant attention. Hot baths, bromides, phenobarbital and local cold and hot applications proved of no value, and finally he had to be given morphine in doses up to 1 grain (0.065 Gm.) per day. This controlled the pain and restlessness, but the priapism was not reduced by this means.

At this point, on the third day, we were called in consultation because of the mental picture. The history from friends brought out that while this was the worst attack ever undergone, the patient had had the first attack of priapism, lasting several hours, during war experiences ten years before. After that he had had similar attacks at intervals, but, while annoying, they were not incapacitating except for the short time during which they existed. However, on the occasion of the present attack he had had erections more or less constantly for a month and they had been much worse for a week. The dreamy delirium mentioned made its appearance, and he had screamed and "been out of his head" the night before.

Examination.—The patient was large and muscular; he was in bed in a dreamy state in which fantasies played a large part. He talked to imaginary people, carrying on a conversation with them in a dreamy way. He was disoriented, but replied to questions readily though in a rather unsatisfactory manner.

The lungs were clear; the heart was sound; the pulse rate was 86; the blood pressure was 115 systolic and 85 diastolic; the arteries were soft. The urine showed a specific gravity of 1.030, a few hyaline casts, ten leukocytes per field, and on one occasion a trace of albumin. The abdomen was normal. The tongue was badly coated, but was protruded straight. The thyroid was normal. The temperature had been high for some days and was now 103.6 F. A blood count showed: white cells, 23,000, mostly polymorphonuclears; red cells, 3,800,000, of normal appearance, and hemoglobin 85 per cent. The prostate was slightly enlarged

but was not tender. There was no history of gonorrhea. The Wassermann reaction was negative. The teeth and skin were normal. He had had no headaches.

The face was drawn a trifle to the right. The right palpebral fissure was narrowed; the pupils were pinpoint (morphine) in size and irregular; nystagmus was present horizontally and vertically; diplopia occurred on looking upward and to the right. The abdominal reflexes were absent; the cremasteric on the left was slight, on the right absent. There was no incontinence or retention of the bladder or rectum. The patellar reflexes were much increased, the right being slightly the stronger; the achilles reflexes were equal and + + +; all reflexes in the upper extremities were + +; there was no Kernig and no Babinski sign. The sole of the right shoe was worn more than the left (the gait could not be tested). The right hand grasp was 140, the left 120; the right leg was stronger than the left. In coordination tests the movements were a trifle clumsy. The legs, which were held flexed at the knees and hips, could be manipulated without discomfort; spontaneous movements were slow and ponderous, but were accurate. Sensibility was normal all over as nearly as could be judged.

The eyegrounds could not be examined.

Priapism.—The patient showed a constant priapism during the two days he was under observation. He also continued to manifest the hysterical state already indicated. He then left the hospital, and no definite information has been secured since, although it is understood that he gradually improved and eventually returned to his occupation, which he has continued to handle successfully.

Diagnosis.—The diagnosis was based on: slight clumsiness; unequal facial innervation; nystagmus; diplopia; absent abdominal reflexes; increased and unequal knee jerks; spasticity and unequal strength in the legs; chronic priapism.

COMMENT

Chronic priapism, with or without remissions, may be considered to be a symptom of cord irritation. It occurs in partial traumatic section of the cord.[4] It is found also in another spastic state, that accompanying the cord lesions in leukemia.[5] It is found also, according to Barker,[6] in "transverse lesions above the lumbar cord, say in the cervical or upper thoracic region, tend to cause continuous erection," but this is an error as this sort of lesion causes congestion only and not true priapism.[4]

As to the location of the lesion or lesions that produce this symptom, one can say: lesions of the region of the centrum genitospinale (from the first to the fourth sacral nerves) produce impotence,[7] hence the lesion must be higher than the sacral region. On the other hand, the dictum that "the precise determination of lesions from symptoms or symptoms from lesions is not possible" [8] was accepted in multiple

4. Bing, Robert: A Compendium of Regional Diagnosis, New York, Rebman Company, 1911, p. 38.

5. Elsner, H. L.: Monographic Medicine, New York, D. Appleton & Company, vol. 6, p. 532.

6. Barker, L. F.: Monographic Medicine, New York, D. Appleton & Company, 1916, vol. 4, p. 280.

7. Bing, Robert: A Compendium of Regional Diagnosis, New York, Rebman Company, 1911, p. 78.

8. Taylor, E. W.: Multiple Sclerosis: The Location of Lesions with Respect to Symptoms, Arch. Neurol. & Psychiat. **7**:561 (May) 1922; Report of Association for Research in Nervous and Mental Disease, New York, Paul B. Hoeber, Inc., 1922, vol. 2, p. 193.

sclerosis by the Association for Research in Nervous and Mental Disease. Accepting this as true we are forced, then, to place the lesion in chronic priapism in the cord higher up than the sacral region.

SUMMARY AND CONCLUSIONS

1. Chronic priapism occurs as an early symptom in multiple sclerosis, and its presence in any nontraumatic case other than leukemia should lead to a suspicion of the presence of this disease.

2. Attention is drawn to the presence of a hysterical state in our case. This condition, too, has been reported but has been considered rare.[1]

3. It is suggested that bizarre symptoms of any kind should arouse a suspicion of the possibility of multiple sclerosis.

INABILITY TO LAUGH AUDIBLY: APHONOGELIA*

MAX LEVIN, M.D., PHILADELPHIA

The inability to laugh audibly is an exceedingly rare neurologic symptom. The authoritative textbooks of Lewandowsky, Oppenheim and Gowers make no mention of it, although Kinnier Wilson said that neurologists have occasion "every now and then" to see patients with this symptom. So far as I can determine, but two cases have been recorded in the literature.

Weisenburg,[1] in 1909, reported a case in a woman, aged 32, who had shown a moderate degree of morbid somnolence since the age of 16. Neurologic examination gave negative results. Treatment, consisting of static electricity, "suggestive therapy" and alterations in the daily routine, was followed by considerable improvement. Weisenburg made the following comment with regard to the laughing function: "A rather interesting feature of her disorder has been that for many years she has never been able to laugh audibly. She is usually of a cheerful, optimistic disposition, and while she would smile, she would not laugh. This has since been improved very much, and she is now able to laugh like any other person."

In 1929, I reported a series of cases of Gélineau's syndrome (narcolepsy).[2] The first case was in a boy, aged 15, who had been having attacks of sleep and of "cataplexy" for one year. The malady manifested itself for the first time on a certain occasion when the patient was about to laugh; suddenly and unexpectedly he found himself able only to smile, and at the same time the leg muscles became momentarily toneless (cataplexy), so that he sank to the floor. Thereafter he was never able to laugh audibly, although his sense of humor was unimpaired. When an occasion for laughter presented itself, his mouth widened into a position appropriate for a smile, but not for a hearty laugh. No sound issued forth. Instead, the patient was aware of a "catching sensation" (or inability to get his

* Submitted for publication, July 16, 1930.

* From the Community Health Center.

1. Weisenburg, T. H.: A Case of Morbid Sleepiness, J. Nerv. & Ment. Dis. **36**:367, 1909.

2. Levin, M.: Narcolepsy (Gélineau's Syndrome) and Other Varieties of Morbid Somnolence, Arch. Neurol. & Psychiat. **22**:1172 (Dec.) 1929.

breath) in his throat, and his eyes blinked. His facial expression at such times was rather grotesque, so that other children said that he was having one of his "goofy spells." Neurologic examination gave negative results. A general physical examination showed a suggestive eunuchoid habitus and orthostatic albuminuria.

To these two cases I shall now add a third.

REPORT OF CASE

History.—An intelligent woman, aged 22, was referred to the Psychiatric Department of the Community Health Center, the problem being that she was the unmarried mother of a child, aged 1 month. The data bearing on the present discussion were: As far back as she could remember, the patient was never able to laugh normally. Even her heartiest laugh was soundless. "It was not a real laugh — more like a smile." When she saw or heard something excruciatingly funny, her lips formed a smile, and tears even came to her eyes, but never was there an audible laugh. "I've always wanted to laugh like other people but just couldn't." Her mother often criticized her by saying, "You never come into the house like other girls with a laugh. You're not lively," "Why don't I ever hear a laugh out of you?" etc. The patient was very sensitive and these criticisms hurt her deeply. When girls came to the house she felt uneasy, knowing that her parents were watching to see whether she was lively and jolly like other girls. For this reason she always enjoyed parties at other girls' homes more than at her own. When parties were given at her own home she could be fairly lively as long as the parents were out of the room, but once they entered she lost all spontaneity. "Nobody knows how I struggled against this shyness." At the age of 18, her shyness became somewhat less apparent, and in its place there appeared certain cynical and caustic qualities, so that people began to think that she was conceited and had an unduly "sharp tongue." This, however, did not prevent her from getting along with her more intimate friends, who realized that back of her caustic remarks there was usually no genuine antagonism.

Examination.—The patient's behavior during the one interview I had with her was normal. She was tall and well developed. The thyroid isthmus was barely palpable. The pulse rate was 72. A brief neurologic examination, including an examination of the pupils, the movements of the face, tongue and outstretched fingers, the gait and the knee-jerks, revealed no abnormalities.

COMMENT

For the inability to execute properly the laughing act I propose the term dysgelia (formed analogously to dysarthria, dysphagia, dysmasesia, etc.). For the specific form of laughing disability common to the three cases cited — the absence of phonation during laughter — the term aphonogelia would seem appropriate.

The cases of abnormality of the laughing function recorded in the literature fall in three main groups:

1. Cases in which the act of laughing is imperfectly performed (dysgelia). In this group are: (*a*) cases of "aphonogelia," and (*b*) cases in which disease of the subcortex, the cerebellum or the facial nerves interferes with the proper innervation of the muscles involved in laughing.

2. Cases in which the act of laughing is qualitatively more or less normal, but appears in response to what is normally an inadequate stimulus. The well known cases of "pathologic laughing" belong in this group.

3. Cases in which the laughing act is qualitatively normal and the stimulus normally adequate, but the laughing act is accompanied by pathologic epiphenomena. In this group are four subdivisions: (*a*) cases of Gélineau's syndrome (narcolepsy), a condition in which there are two characteristic symptoms, attacks of transitory muscular tonelessness on laughing or other emotional display (cataplexy), and attacks of sleep; more than 100 cases of this syndrome may be found in the literature; (*b*) cases of cataplexy unaccompanied by attacks of sleep, about a dozen of which have been reported; (*c*) cases of what Oppenheim called "Lachschlag." In this condition when the patient begins to laugh, he falls unconscious, in contrast to the cataplectic patient who is toneless but conscious during the attack; (*d*) cases in which laughter precipitates an epileptic attack.

It is worth emphasizing that no disorder in laughing constitutes a disease in itself. It is always best looked on as merely a symptom of some underlying condition. That the patient with aphonogelia herein reported had no other signs or symptoms of disease does not invalidate this point of view.

Practically nothing is known regarding the mechanism responsible for the occurrence of aphonogelia.

SUMMARY

Report is made of a case of "aphonogelia," or the inability to laugh audibly. The patient showed no other symptoms or signs of disease. The literature contains two other cases presenting this symptom: One patient had attacks of sleep, and the other had Gélineau's syndrome (attacks of sleep and cataplexy).

QUANTITATIVE PUPILLARY LIGHT REFLEX*

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Neuro-Ophthalmologist, Philadelphia General Hospital; Assistant Surgeon, Wills Hospital

The instrument here presented is one that measures accurately the intensity of light, designated in candle power, and is intended as an aid in determining quantitatively the pupillary response to light. In is an improvement on a similar instrument, containing only a hand battery, which was exhibited to the Section on Ophthalmology of the College of Physicians of Philadelphia on April 19, 1928.

That apparatus was exhibited, not for its accuracy, but for the principle involved in designating the candle power of light of three intensities, one a minimum, another a medium and the third a maximum.

I was aware that that instrument, containing a dry battery, would not always function accurately, because the dry battery would depreciate in a short time. Practical use of the instrument proved, however, that it could be used in the office for a period of two weeks before any serious loss in the intensity of the light would take place.

It was appreciated that batteries of different makes, when new, produce varying intensities of light. The rheostat, however, in this same instrument was so arranged

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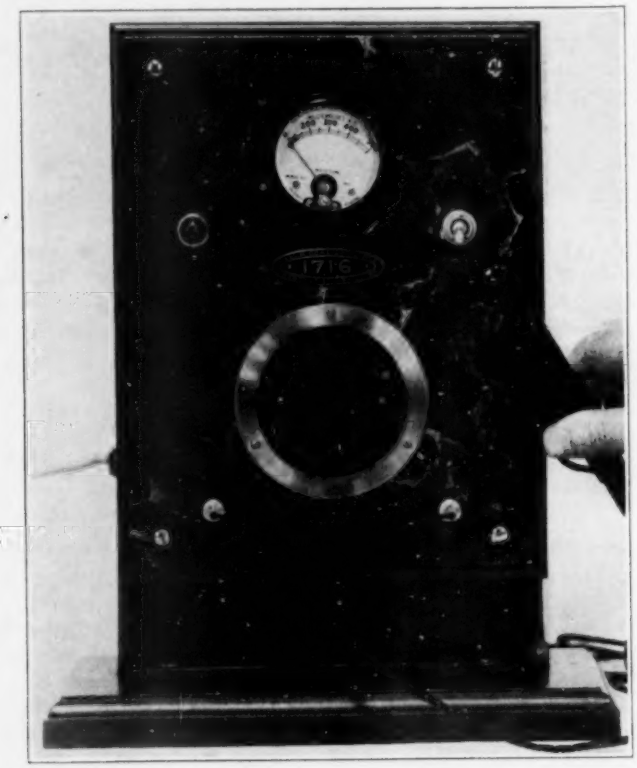
* Read before the Philadelphia Neurological Society, May 23, 1930.

that it would divide the candle power fairly evenly so that, for comparative purposes, it would be of practical value in measuring the response of the pupil to different intensities of light.

In order to avoid the inaccuracies occurring in the use of the device containing a hand battery, I have devised the instrument here described for the same purpose; but it is electrified by the house current and equipped with a rheostat and a milliampere meter, which will give the exact number of milliamperes delivered to the electric bulb, the light of which can be measured in candle power by a Westinghouse foot candle instrument. In this instrument there is little or no



Electric instrument having milliampere meter and rheostat, to which is attached a cord and handle. The head of the handle contains a 40 diopter lens.

variation of the intensity of the light used for the purpose of determining the pupillary light reflex.

I have designated three intensities of light: 10 foot candle power illumination at 1 inch (2.5 cm.) from the focusing lens, using a current of 250 ma.; a second unit of 20 foot candle power, using 275 ma., and another unit of 40 foot candle power, using 300 ma. The electric bulb is a mazda of 2.5 volts. When the glass of the bulb becomes darkened by use it may be readily replaced.

A light of less intensity than 10 foot candles would cause contraction of the pupil, it being known that even ultraviolet light may cause contraction. But the

luminosity must be sufficient for the observer to see the reaction of the pupils. The ordinary subdued daylight, or artificial light in the office, should not be greater than 4 candle power at the position of the patient's eyes. Such a light would be equivalent to that in the usual artificially illuminated office, examining room or ward in a hospital.

In arriving at an arbitrary minimum, consideration was given to the fact that deeply pigmented irides are less responsive to light than those less pigmented. Persons with a high degree of myopia are more sensitive to light than are those with hyperopia, but the pupils of the former are less active.

It would be impossible to create a standard of the reaction to light for all types of illumination in the examining room. I have arrived, therefore, at the conclusion that a 10 candle power light will produce a pupillary reaction in the various grades of normal eyes.

When the pupil reacts to such a light, it may be designated as plus 3. If a 20 candle power light is required to produce a contraction it is designated as plus 2. If a 40 candle power light is required, the pupillary response is feeble and regarded as plus 1. When no response is obtained by the 40 candle power light, one may, for comparative purposes, regard the pupil as inactive to light.

It is true that a response may be obtained sometimes when using a light of high intensity even in the presence of optic atrophy. It is even claimed that the musculature of the iris, being derived from the ectoderm, may of itself respond directly to the stimulation of light.

These extraordinary factors, however, do not enter into the attempt to measure the comparative response of the pupil to varying intensities of light. From the standpoint of the neuro-ophthalmologist, the pupil that fails to react to a stimulus of 40 candle power may be regarded as inactive to light.

I have failed intentionally to include in this brief paper any comment on the nervous mechanism concerned in the pupillary light reflex. It is stated that the paths subserving the sphincter muscle are alone concerned with the response to light, and that their course is largely hypothetic.

It is well known that certain types of pupillary reaction are met under varying pathologic conditions, that the visual pathways and the pathways of light are in close association with each other and that the disturbances of the brain caused by vascular diseases, new growths and general systemic diseases cause variations of the pupillary light reflex.

One knows that there is not always an abrupt dividing line between complete loss of light reflex and full activity of the sphincter muscle. The grades between these two extremes may be easily designated by the instrument that I have described here. It will serve as a means of definite record and comparison, so that the observer may be able to know whether the approximate measure of the light reflex is diminishing or fixed or, once lost, if it is recovering. With the use of this instrument, no differences of opinion will arise as to whether a pupil is active or inactive to light. The device shown here is merely a laboratory specimen which may, by experiment, be made more compact for general use.

ABSTRACT OF DISCUSSION

Dr. Charles K. Mills: The instrument shown by Dr. Lehrfeld is of real importance. From my own experience I know that it is desirable to have some practical apparatus to measure the amount of light reflex. The appliance is ingenious and on the whole appears to represent a convenient apparatus for demonstrating the amount of light reflex.

DR. MAXWELL LANGDON: I think that Dr. Lehrfeld's instrument is most interesting and ingenious, but I am not sure that it will become the method of general use. There are many normal factors that have a wide variation—the congenital size of the pupil, the error of refraction and the development of the sphincter pupillae. All of these must make a tremendous difference in pupillary reactions.

It seems to me that a good clinical method is to test the pupillary reactions with a good skylight from a north window, or from an east or west window in which the sun is not shining, and if there is no reaction, to focus the artificial light on the eye through a condensing lamp. A record can then be made of whether a reaction was obtained with the diffuse or the condensed light. The method, of course, is much cruder than Dr. Lehrfeld's, but it seems to me to be a good clinical method.

DR. LOUIS LEHRFELD: In reply to the query regarding the examination of patients in the dark room, the instrument was devised for practical examination of the patient at the bedside and in the office. In response to Dr. Langdon, the apparatus looks complicated, but it can be condensed. I believe that we should continue to look for scientific accuracy and that this instrument will serve in that direction.

A PRIMARY MALIGNANT TUMOR OF THE SOLAR PLEXUS*

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A case of rare primary tumor of the solar plexus, which presented an unusual clinical picture as well as a strange and striking morphology, is presented.

CASE REPORT

History.—Mrs. O., aged 60, a housewife, the mother of two healthy, grown children, entered the hospital complaining of pain in the right lower quadrant of the abdomen which had been present for about four months. She slept poorly and had a poor appetite; the bowels were irregular (diarrhea alternating with constipation), and she had lost about 20 pounds (9 Kg.). Curettage had been performed shortly after the first childbirth; bronchopneumonia had occurred twice and erysipelas once, and two years before, several papillomas had been removed from the feet. She had also had some form of arthritis for twenty years.

Physical Examination.—The uterus was tender, and there was some tenderness in the abdomen to the right and above the umbilicus. Gastric succussion also was present. Roentgen examination showed a normal duodenal cap and normal emptying time of the stomach. There was, however, a constant and definite filling defect of the duodenal cap; the barium passed through rapidly, suggesting some duodenal lesion.

Course.—The patient was given a Lenhartz diet for duodenal ulcer. She did well for a few days and was allowed to go home, but soon shifting tenderness

* Submitted for publication, July 23, 1930.

* From St. Luke's Hospital.

in the abdomen, intermittent diarrhea, occasional pain after eating and, later, abdominal distention and shooting pains in the legs and hands developed.

Operation.—She returned to the hospital, and an exploratory operation was performed by one of us (E. L. M.) two days after the second entry to the hospital. An immobile retroperitoneal tumor, lying to the right and above the head of the pancreas, was found. This was left untouched. It had, in the gross, the invasive characteristics of a sarcoma, and was so diagnosed. A normal appendix was removed, and the incision closed.

Postoperative Course.—The patient was put to bed in a good condition, but did not come out of the anesthetic, appearing to remain in a state of shock, although the pulse was good and the temperature was normal. Throughout the entire postoperative course, until death, she at no time regained complete consciousness, but gradually fever developed and she died ten days after the operation. The patient could be roused by questions but apparently could not exert enough energy to control her activities. Respiration was shallow, but nothing was found in the lungs or heart to account for this depression. The abdomen was soft, except for one area of muscle tension located at the tenth right costal cartilage.

An interesting observation was made at this time by one of us (G. H. H.), who stated, "Something seems to be inhibiting the vegetative nervous system, and her body is apparently functioning only through the effort of will power (cerebrospinal system)."

Necropsy.—There were no discoveries of interest, except those related to the tumor. In the abdomen, immediately posterior to the duodenum and elevating it, a rather well fixed mass was found which measured about 6 cm. in diameter. On careful dissection it was seen to be an infiltrating fibrocellular tumor, bounded on the right and left by the suprarenal gland and the kidney, and posteriorly by the aorta, which it partially surrounded. It extended about 3 cm. below the bifurcation of the renal arteries. The celiac artery ran through the capsule at the upper border, and a portion of both the right and the left celiac ganglia could be dissected loose from the tumor into which their ramifications ran and were lost. It was apparently springing from the masses of plexuses composing the solar group, and only the two ganglia mentioned were definitely made out. The fibers of all the plexuses ran directly into the tumor and were lost. The tumor mass was fairly well capsulated, firm and semielastic in consistency, spheroidal in shape, and on cross-section presented a grayish cellularity resembling sarcoma. Many large grayish-green areas of semigranularity suggesting necrosis were seen on cross-section; some of them measured as much as 25 mm. in diameter. These were poorly outlined and blended into the surrounding rather firm, grayish-white tumor tissue about them. There was no involvement of the adjacent lymph nodes, and a careful examination of the remainder of the body failed to reveal any evidence of regional or remote dissemination of this mysterious growth.

Histologic Observations.—Drs. Arthur E. Hertzler, James Ewing and Francis C. Wood helped us in classifying this unusual neoplasm. Dr. Wood was uncertain as to the exact histogenesis, but thought that it might possibly have sprung from retroperitoneal lymph gland reticulum. Drs. Hertzler and Ewing independently concurred with us in thinking that it was of neurogenic origin. This conclusion was based on the following points: absence of primary source (aside from the solar plexus); lack of regional lymph node involvement and peculiar morphology. Hence, we believed that it was a primary tumor of sympathetic nerve ancestry.

The tumor was fixed in formaldehyde and Zenker's fluid. Hematoxylin and eosin, van Gieson, Mallory's phostophotungstic acid and Bielschowsky's staining methods were employed in attempting to classify the tumor histologically.

Under a low power magnification the sections showed large areas of necrosis which took a pinkish, semigranular stain with the hematoxylin-eosin stain. The main mass of the nonnecrotic tumor was made up of a cellular type of growth, the cells of which were for the most part small, round and spindle-shaped. The

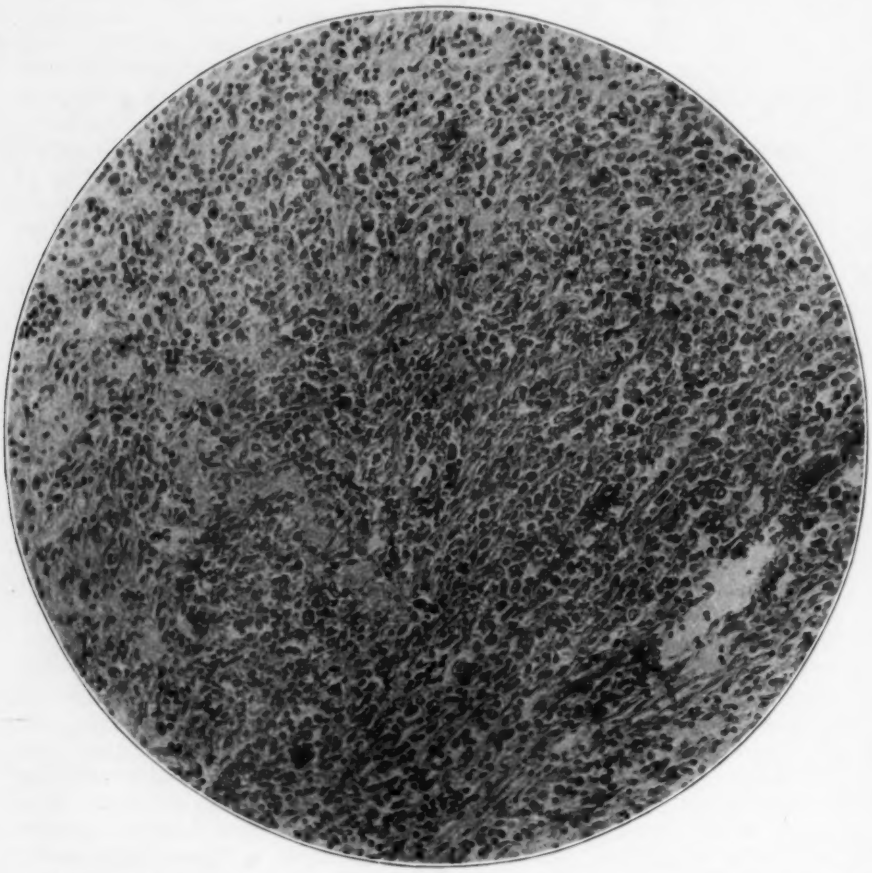

Fig. 1.—Low power photomicrograph of tumor, showing sarcomatous arrangement of cells; × 160.

stroma varied in amount. In some areas it was scanty; in others, abundant. Running throughout the sections small nerve fibers and small bundles of nerve fibers were seen, many of them showing degenerative changes. Under higher magnification the cells forming the tumor parenchyma varied considerably in size and shape. They were found to be invading the capsule on all sides, and in the body of the tumor the cells lay directly on the endothelial lining of young, poorly developed vascular channels. Most of the tumor was composed of irregular round and spindle-form cells, many of the latter having definite cytoplasmic

"tails" which strongly suggested a neurogenic origin. No definite large ganglion cell forms were seen, but some large cells with atypical nuclear structures were occasionally encountered. Mitotic figures were not numerous but could be found in many fields. Irregular, small, dense, pyknotic nuclei, with little or no cytoplasm, were also seen. There was a low grade, scattered infiltration of round cells in some areas. In places the tumor showed considerable vascularity, while in others it was densely fibrotic and avascular. This was particularly true adjacent to the

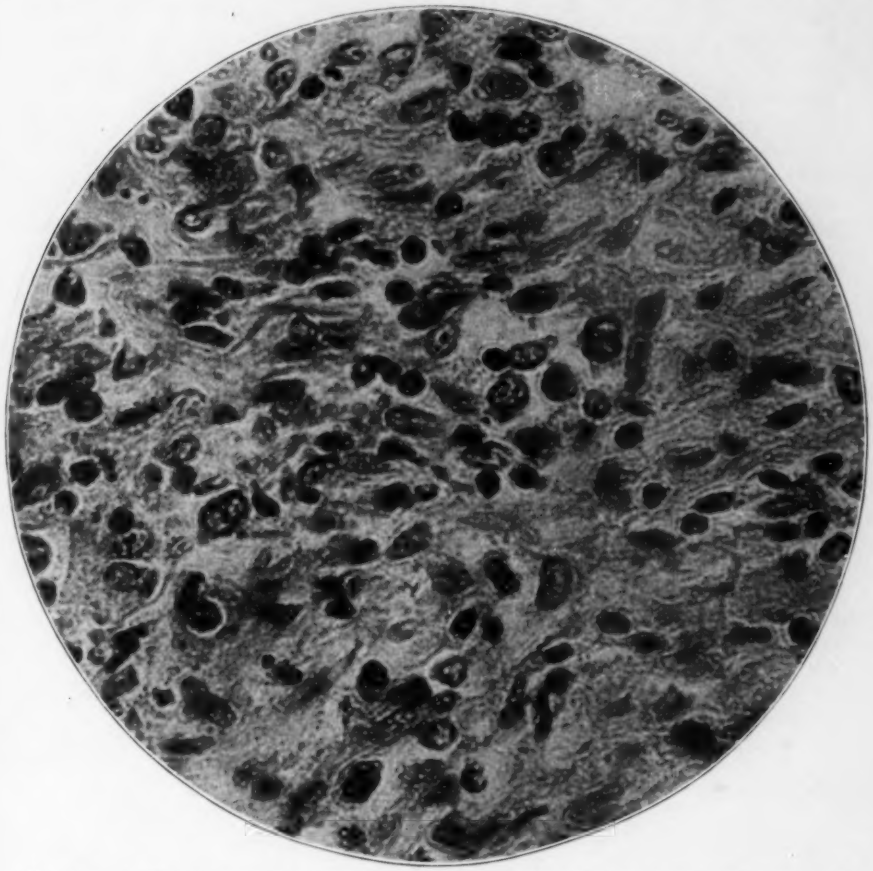

Fig. 2.—High power photomicrograph, showing variation in cell type and cytoplasmic processes; × 700.

capsule. A diagnosis of neurosarcoma was made after considerable deliberation and study with differential stains.

The first impression gained from a cursory examination of the microscopic sections was that we were dealing with an undifferentiated sarcoma. However, the Bielschowsky, phosphotungstic acid and van Gieson stains revealed small bundles and strands of broken and wavy neurofibrils and many of the cells showed definite "tails," These observations made us feel sure that we were dealing with a neoplasm of nerve tissue origin. Classification of the type of tumor, even if of embryonal

nerve tissue origin, is difficult. In the more restricted sense we did not believe that it was a neuroblastoma since neither ganglion cell forms nor "rosets" were seen.

The gross anatomy showed a definite and unmistakable involvement of the solar plexus with the ganglia buried within it. The majority of the neuroblastomas have been described as arising from the medullary portion of the suprarenal gland; hence, we carefully eliminated any such eventuality in regard to our neoplasm.

Fig. 3.—High power photomicrograph, showing cytoplasmic "tails." Special stains showed these to be probably of nerve origin; × 1,200.

CLINICAL COMMENT

The observations seem to check with the long clinical history of some vague upset in the vegetative nervous system. By some observers the patient had been considered neurasthenic; no adequate cause for her complaints was found. Everything she did seemed to be at the cost of great mental effort.

It is interesting to speculate as to whether the tumor might have had some influence in the production of the intermittent diarrhea, nausea and vomiting, the pains in the legs and the so-called attacks of arthritis.

Diagnosis.—Before the operation, a duodenal ulcer was practically ruled out because of the inconstancy of the symptoms. We thought that there was probably extrinsic pressure on the stomach and pylorus but no intrinsic lesions. The appendix, the ureter and the kidney were excluded because of the inconstancy of the symptoms, and the other usual signs and symptoms were not present. In looking over the history one is struck by the inconstancy of the reference to the pain in the back over the liver region. The patient referred to the pain as being under the shoulder blade. Naturally this made us think of involvement of the gallbladder, but we could get no other evidence of such trouble. The presence of the tumor had in no way affected sugar metabolism, nor were any stigmas of suprarenal involvement present.

Wahl,[1] Hook[2] and Wassmund[3] have given comprehensive reviews of the tumors arising from the sympathetic nervous system. Singer[4] reported two cases of primary involvement of the celiac plexus in which both were removed surgically with apparent recovery. How long these two patients remained well is not mentioned. Wahl collected sixteen cases of retroperitoneal ganglioneuroma arising from the sympathetic nervous system outside the suprarenal gland. Only one was definitely described as springing from the celiac plexus. He also tabulated twenty-five malignant neuroblastomas, none of which was spoken of as arising from the solar plexus. Eleven of those arose in the medulla of the suprarenal gland.

These malignant forms have been described as arising in the brain, gasserian ganglion, sympathetic ganglion of the neck, broad ligament, sacral sympathetic and other regions; in fact, in almost every region in which there is an abundant collection of sympathetic nerves and ganglia. Many retroperitoneal tumors reported as sarcomas might, on careful analysis, be found to be of primary nerve tissue origin.

SUMMARY AND CONCLUSION

1. An unusual primary malignant tumor arising from the solar plexus is reported.
2. The clinical picture was both unusual and striking.
3. The type of tumor does not fit in morphologically with any that we have been able to find.

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1. Wahl, H. R.: J. M. Research **30**:205, 1914.
2. Hook: Frankfurt. Ztschr. f. Path. **7**:135, 1911.
3. Wassmund: Virchows Arch. f. path. Anat. **226**:319, 1919.
4. Singer, B.: Deutsche Arch. f. Nervenh. **92**:240, 1926.

News and Comment

EIGHTH ANNUAL MEETING OF AMERICAN ORTHOPSYCHIATRIC ASSOCIATION

The Eighth Annual Meeting of the American Orthopsychiatric Association will be held at the Hotel Pennsylvania, New York, Feb. 20 and 21, 1931.

NATIONAL COMMITTEE FOR MENTAL HYGIENE

At the twenty-first anniversary meeting of the National Committee for Mental Hygiene in New York on Nov. 13, 1930, the development of a nation-wide program of research in cooperation with American universities was announced as the next step in the extension of the activities of the Committee. Dr. C. M. Hincks, founder and medical director of the Canadian National Committee for Mental Hygiene and a vice president of the International Committee for Mental Hygiene, was introduced as the new general director of the organization, succeeding Dr. Frankwood E. Williams, who will retire on Jan. 1, 1931, after fourteen years of service with the Committee.

A MEDICAL CENTER FOR URUGUAY

The Government of Uruguay has approved a plan for a medical center in Montevideo which is based on the practices and development of the medical center of New York. The new center will be patterned exactly after that in New York and the buildings will exceed those in New York both in size and in height (twenty-three stories). Dr. G. Charles Burlingame, executive officer of the New York Medical Center during its planning and construction, has acted as special consultant to the Uruguayan government in the preparation of the plans. Included in the program is provision for a psychiatric institute, the first of its kind in South America.

Obituary

HERMON CAMP GORDINIER, M.D.
1864-1930

Dr. Hermon Camp Gordinier, an acknowledged master in the domain of the medical sciences, passed away at his home in Troy, N. Y., on Sunday, Oct. 19, 1930, after a prolonged illness. Stricken in 1925, at the age of 61, with thrombosis of a branch of the left coronary artery, he never fully recovered. Prematurely crippled, Dr. Gordinier stoically faced the demoralizing evidence of his increasing invalidism and the cruel ordeal of repeatedly enforced inactivity. One of Troy's most illustrious sons, one of New York State's foremost medical authorities, and, it is believed, one of the nation's most brilliant clinicians and most remarkable teachers, Dr. Gordinier's demise brought a keen realization of its staggering loss to the extensive community to which he had ministered so ably and faithfully during his long and honorable career.

Dr. Gordinier was born at Troy, N. Y., on May 21, 1864. He attended the public schools in Troy and received his medical education at the Albany Medical College from which he was graduated in 1886. He then pursued postgraduate studies at the New York Polyclinic School (1887-1888) and at the universities of Prague, Vienna and Berlin (1889-1890).

After his return from Europe, Dr. Gordinier became connected with the Albany Medical College as well as with various hospitals of his native city, being appointed physician and neurologist to the Samaritan Hospital and to the Marshall Sanitorium in Troy, and likewise serving in later years as consulting physician and neurologist to the Mary McClellan Hospital in Cambridge, N. Y. During the war he was chief medical consultant to the U. S. Army Draft Boards of Rensselaer, Washington and Albany counties.

Gifted with a robust constitution, an exceptionally keen mind and an unlimited capacity for work, Dr. Gordinier gave evidence, from the moment of his advent in medical practice, of a prodigious momentum and a predestination to scientific fame and professional leadership. Constantly in quest of knowledge, he literally devoured both classic works and current medical publications and, thanks to a marvelous memory, stored a huge fund of available information. His ability to furnish offhand a reference or useful lead was astonishing. Although he exhibited, even from student days, a decided preference for specializing

in the treatment of diseases of the nervous system, and published, more than thirty years ago, an epochal textbook on the anatomy of the nervous system,[1] at that time the only complete and comprehensive treatise on the subject in the English language, Dr. Gordinier's scientific leanings and interests carried him practically into every phase of medicine, and he rapidly acquired an enviable reputation both as a neurologist and as a general clinician. In great demand as a consultant throughout central and northern New York as well as in the adjoining sections of Massachusetts, Vermont and New Hampshire, Dr. Gordinier's life was an endless display of service and self-sacrifice.

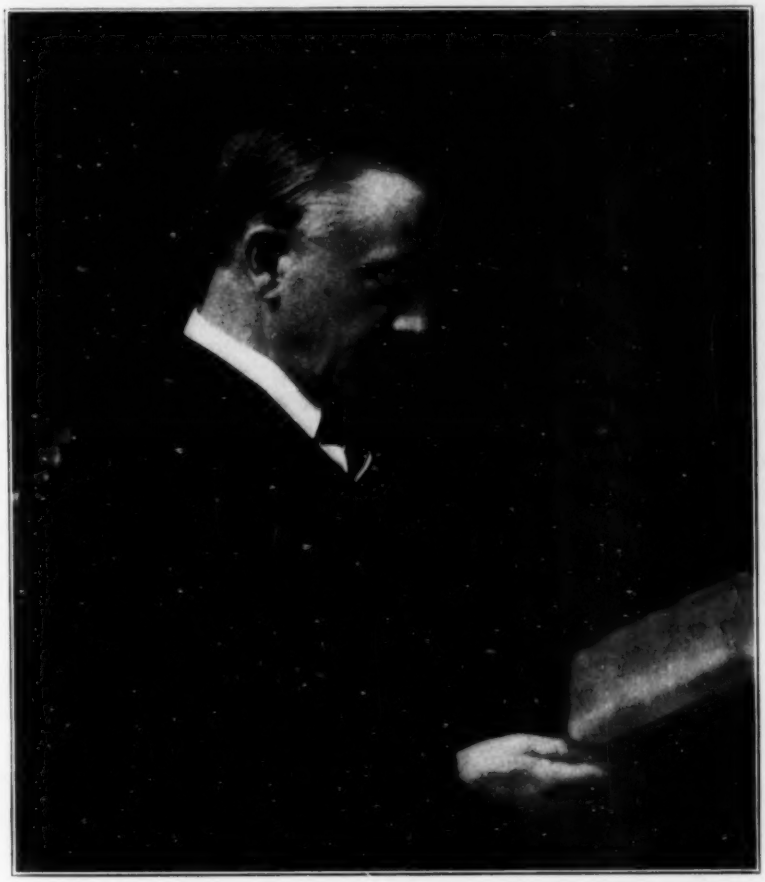
During a period of forty years, Dr. Gordinier was actively identified with the teaching staff of his alma mater, Albany Medical College (Medical Department of Union University). After holding minor positions for a short time, he was appointed as professor of physiology and of anatomy of the nervous system, and subsequently (1915) as professor of medicine. With untiring zeal and devotion he served the school and brought to it the fertile influence of his scholarly equipment and exemplary courage. His share in shaping the future destinies of the institution, in perfecting its educational standards and in enabling it, despite its humble size and inadequate endowment, to attain and maintain the highest degree of efficiency, is incalculable. If Dr. Gordinier gained wide recognition as a scientist and a clinician, it was in his capacity as a teacher that he gave the full measure of his talent and ability and achieved his greatest triumph. Few men have brought to the clinical amphitheater a more conscientious preparation, a more unassailable document or a more dynamic and convincing presentation. With a magnetic personality, a countenance glowing with energy and intelligence, a melodious and resonant voice and exquisite diction, Dr. Gordinier was the embodiment of professorial prestige and distinction. An educator of the first magnitude, he was eminently qualified to fill the chair of medicine in any medical school. No one held a warmer place in the hearts of his pupils and associates. He worked for them and with them, kindling their enthusiasm, encouraging their original trends, guiding their researches with the utmost patience and solicitude and giving them the invaluable aid of his vast erudition and experience.

As an author, Dr. Gordinier has left an imperishable record in the annals of American medicine. The impressive list of his publications,[2] comprising more than seventy titles, is an eloquent index of his unusual voltage. A critical survey of the content of Dr. Gordinier's bibliography reveals not only the wide range of his scientific pursuits, but the highly

1. Gordinier: Gross and Minute Anatomy of the Central Nervous System, Philadelphia, P. Blakiston's Son & Company, 1899.

2. Gordinier, in Semi-Centennial Anniversary Volume of the American Neurological Association 1875-1924, Albany, N. Y., Boyd Printing Company.

significant fact that purely clinical studies were few while clinico-pathologic contributions, many of them of the highest merit and originality, constituted the overwhelming majority. A fitting appraisal of his accomplishment is practically impossible, for he delved into almost every major problem in medicine. Although necessarily obsolete in some of its chapters, Dr. Gordinier's textbook on the anatomy of the



HERMON CAMP GORDINIER, M.D.
1864-1930

nervous system nevertheless stands as a monument to the part he unquestionably played in the emancipation and assertion of American authorship. An ardent supporter of the classic doctrine of cerebral localization and a firm believer in the rigid delimitation of functional centers in the cerebral cortex, Dr. Gordinier fought valiantly for the existence of a separate cortical center for writing at the base of the

second frontal convolution of the left hemisphere in right-handed persons. One of the first in this country adequately to describe the clinical features and underlying pathology of acute ascending paralysis of the Landry type, reports of cases of which he published on several occasions, Dr. Gordinier apparently continued to envisage this syndrome as a well defined disease entity. He reported a number of unusual and instructive cases of tumor of the brain and contributed valuable articles on multiple neuritis, syringomyelia and occlusion of the posterior inferior cerebellar artery. Outside the realm of neurology, Dr. Gordinier's chief interests were centered on diseases of the cardiovascular system, the blood and blood-forming organs and the endocrine glands, and accordingly one finds in his collected papers a large number of illuminating articles on aneurysm of the thoracic aorta, malignant endocarditis, lymphatic and myelogenous leukemia, myxedema and sporadic cretinism, acromegaly and other closely related disorders. In 1889, he published "A Case of Sclerodactylia with Diffuse Scleroderma," which was the first recorded case in America.[3]

Throughout his writings Dr. Gordinier betrays the predominating influence of his pedagogic instincts, and, without neglecting correctness of form or choice of expression, he is more concerned with descriptive accuracy and clarity of exposition than with elegance of style. Invariably prefaced by a loyal account of the legacies of earlier investigators, Dr. Gordinier's subject is presented with methodical order and precision, clinical and pathologic data are exhaustively detailed, functional deficits are judiciously correlated with anatomic localization, differential diagnosis is ably discussed, and, when indicated, prognostic and therapeutic considerations are scrupulously evaluated. In fine, Dr. Gordinier's articles bear the stamp of scientific thoroughness and practical utility.

Honored on innumerable occasions by medical societies and organizations throughout the state, Dr. Gordinier received in 1896 the degree of Master of Arts from Williams College and, in June, 1930, Union University conferred on him the degree of Doctor of Science in recognition of his notable contributions to medical science. Dr. Gordinier was admitted to the American Neurological Association in 1898. He was vice-president in 1921 and served as councillor. He was also a member of the Rensselaer County Medical and New York State Medical Societies, American Medical Association, Association of American Physicians, American Therapeutic Society, Aesculapian Club of Albany and New York Academy of Medicine.

Viewed in the more intimate atmosphere of private life, Dr. Gordinier was a man of rare charm, unfailing courtesy and sympathetic nature. Modest to a fault, generous in his judgment of others, cautious

3. Gordinier: Am. J. M. Sc. **97**:15, 1889.

in his deliberations and proverbially just in his decisions, his reputation of professional and civic integrity was a byword. Profoundly devoted to the welfare and happiness of his family and knowing no greater delight than the companionship of his children, Dr. Gordinier never allowed personal cares or concerns to submerge his responsibilities as a citizen, and took a keen interest in all matters that pertained to the industrial and educational growth of his native city. A standard-bearer of the highest moral and ethical principles, he enjoyed the unalienable respect, confidence and esteem of his patients, colleagues and fellow citizens. A great lover of outdoor life, Dr. Gordinier eagerly seized every opportunity to take long cross-country jaunts or to pursue his well known hobby of studying the flora of whatever territory he happened to traverse. A botanist of no mean merit, he accumulated an extensive collection of dried plants and flowers and incorporated his observations in at least two noteworthy monographs. Practically every fall, he managed to slip away and indulge in his favorite sport of hunting. His game was mainly woodcock and partridge, and he was considered an excellent shot. Very fond of good music and the legitimate stage, but above all an omnivorous reader, it may be said of Dr. Gordinier that to the very last he remained a tireless, enthusiastic and insatiable student.

In 1894, Dr. Gordinier married Miss Alice Beattie. They had five children. He is survived by two daughters, Miss Hermione Gordinier and Mrs. George P. Ide, 2nd, both of Troy, and a son, Hermon C. Gordinier, Jr., a medical student at Johns Hopkins University, Baltimore.

Abstracts from Current Literature

HEADACHES IN THE SYNDROMES OF THE SPHENOIDAL FISSURE AND THE APEX OF THE ORBIT. HENRI ROGER, Rev. d'oto-neuro-opht. **7**:581 (Oct.) 1929.

The syndrome of the sphenoidal fissure is due to a lesion, developing in the periphery of the fissure or the adjacent area, which compresses the third, fourth, sixth and the first branch of the fifth nerves and the ophthalmic veins that traverse it. It causes paralysis, successively, of the motor oculi (beginning with ptosis and finally involving the pupil), the patheticus and the external motor oculi, which results in complete immobility of the globe; through involvement of the ophthalmic branch there occurs hypesthesia of the supra-orbital region and of the cornea. To this is added a more or less marked exophthalmos. When the lesion invades the contiguous optic canal there is added to the foregoing the syndrome of the apex of the orbit, characterized by amaurosis without change in the eyegrounds. Déjean divides the paralytic syndromes of the summit of the orbit into four groups: ophthalmoplegia sensorio-sensitivomotor, ophthalmoplegia sensitivomotor, ophthalmoplegia motor total and ophthalmoplegia incomplete.

This discussion is based on the observations in eight cases, four of which have previously been reported. Five are cases of the syndrome of the sphenoidal fissure and three of the syndrome of the apex of the orbit.

According to the classic descriptions, the syndrome of the sphenoidal fissure begins with paralytic signs, is followed by exophthalmos and complete fixation of the globe and finally by disturbances of sensation in the domain of the ophthalmic branch. Later, when the apex of the orbit is involved, lowered vision without modification of the papilla occurs. The progress is rapid; one case showed paralysis of the third nerve in four days, one in several days, while two cases required some weeks for the complete evolution. There is a relatively less marked involvement of the intrinsic than of the extrinsic musculature. The dogma that the third nerve is involved before the sixth is not always true, one case showing a horizontal diplopia two weeks before the third nerve was affected. Only two cases showed exophthalmos. In one case edema of the lower lid was observed. Although the ophthalmic veins are compressed, collateral circulation is established through the angular vein. All three cases of the syndrome of the apex of the orbit showed lowered vision without changes in the eyegrounds.

In some cases roentgenography of the sphenoidal fissure may reveal interesting lesions. In one case there was opacity of the orbit at the level of the fissure and in another an enlargement and malformation of the fissure, together with absence of the bony point separating the fissure from the optic canal. (Vision was not disturbed in this case.) In the other cases, in spite of undoubted lesions, the two sides were alike in the films.

The absence of disturbances in the other cranial nerves and in the general nervous system is important. In one case, the sphenoidal fissure of the opposite side became involved by the neoplasm some days before death.

Headache was present in all of the cases. Indeed it dominated the picture for a long time. Most of the patients were not troubled by the diplopia or the unilaterally lowered vision because of the ptosis; but they strenuously demanded relief from the atrocious headaches. In all of the patients, this was the first symptom observed. According to ophthalmologists, the absence of pain often causes patients to delay seeking help until the paralysis is complete. The suggestion is made that it is not impossible that there are two types of the disease, painful and nonpainful, and that patients with the former are apt to consult the neurologist and the latter the oculist. The seat of the pain is most often temporal or parietal rather than frontal or intra-orbital. In some cases the internal nasal nerve is more painful than the supra-orbital. The pain is apt to begin in the periphery of the supra-orbital; in one case the pain remained in the posterior parietal region for several weeks; in another it began near the vertex and then descended toward the forehead and root of the nose. This pain begins as an aching and pro-

gresses in most cases, but sometimes the onset is sudden. In all of the cases, neuralgia preceded the ocular troubles by several days or weeks, and was violent; one robust patient cried out and, in despair, would strike his head against the wall. Its duration varies with the case; the majority of the patients suffered during the whole course of the evolution of the syndrome. In one case, lying on the side affected increased the pain, and strong pressure over the supra-orbital notch temporarily relieved it. Pressure over this point is rarely painful. Hypesthesia, especially in the domain of the supra-orbital nerve, was noted and exceptionally may extend to the territory of the internal nasal, the superior maxillary or even the inferior maxillary nerve. Corneal hypesthesia is frequent, but complete anesthesia with disappearance of the corneal reflex is rare.

Slight vertigo, even with nausea, was noted in three cases. One case merits special mention. This patient suffered violent supra-orbital pain and kept the ptosed eye bandaged; she resisted all attempts to examine the eye. Each time the lid was raised, vertigo with pallor and anxiety were very distressing, although the examination was made in a dim light and the unaffected eye was kept closed. The vertigo was less severe in a dark room. It was attributed to a special vestibular reflex, due to a dazzling of the affected eye, the pupil of which was dilated, by a relatively feeble illumination.

In its beginning, the headache must be differentiated from other unilateral supra-orbital headaches. The essential characteristics are: onset in the parietal or temporal rather than in the frontal region ("distal" neuralgia); continuous, but with periods of exacerbations, often nocturnal, of a particularly intense and rebellious type; supra-orbital and corneal hypesthesia. Essential facial neuralgia and inflammations of the nasal sinus are easily ruled out by appropriate examinations. The neuralgias following zona ophthalmica are distinguished by the zosterian eruption or the cicatrices remaining. A lesion of the parietal bone may be simulated by cases in which the neuralgia is confined to this region. When it is determined that the pain is in the territory of the supra-orbital distribution, attention will be directed to the nerve in the fissure ("distal" neuralgia).

Certain cases of ocular paralyses caused by lesions situated in the orbit or near the cavernous sinus may confuse the diagnosis. Intra-orbital lesions cause a deep pain, arising spontaneously or from pressure on the globe, considerable nonpulsating exophthalmos and edema of the upper lid. These are much less marked in lesions of the fissure.

In the etiologic diagnosis, three things are discussed: traumatism, syphilis and cancer. In the first, the anamnesis is of determining value. Supra-orbital neuralgia is exceptional from trauma though puncture wounds in the orbit may cause neuralgia by irritation or analgesia by dividing it. In syphilis, which is the most frequent cause of the disease and the most amenable to treatment, there is either gumma or, more often, an osteoperiostitis of the sphenoid. It is a curious fact, however, that the history may be irrelevant and the Wassermann reaction may be negative. In three of the author's cases the Wassermann tests of the blood and spinal fluid were negative, and yet antisyphilitic treatment was efficacious. On the other hand, in one case, due to a neoplasm, the Wassermann reaction was partially positive in the spinal fluid but negative in the blood. Cancer is the cause next in frequency; occasionally it is primary in the orbit or nasopharynx, but more often it is secondary. In one of the cases it was probably primary, in one it was secondary to cancer of the breast, in another to hypernephroma and in still another, there were multiple epitheliomas in the skin, patella and cervical cord.

Guichard reported a case in which trauma, cancer and traumatism were the etiologic factors. In all cases of neoplastic etiology the headache is violent, continuous and of long duration, but this is not enough to differentiate cancer from syphilis. Even in cancer, the neuralgia may be ameliorated toward the end by destruction of the nerve by the neoplasm.

Treatment must be based on the etiology. In cases of doubt, antisyphilitic treatment often succeeds astonishingly well. Roentgen therapy may be tried in secondary cancers or in the primary ones that are beyond the resources of surgical intervention.

DENNIS, Colorado Springs, Colo.

THE PATHOLOGY OF ARSPHENAMINE INTOXICATION OF THE NERVOUS SYSTEM. EUGEN POLLAK and GUSTAV RIEHL, JR., Jahrb. f. Psychiat. u. Neurol. **47**: 100, 1930.

According to Pollak and Riehl, the toxic effects of arsphenamine on the brain may give rise to four types of lesions. 1. The typical picture of purpura. In this type, they believe, there occurs a primary injury to the endothelium of the cerebral blood vessels. The injured endothelium leads to a permeability of the vessel wall which permits an extravasation of the elements of the blood into the surrounding tissues. The microscopic picture of the smallest vessels (capillaries and precapillaries) indicates a primary insufficiency of the innermost coat of the vessels as prerequisite for cerebral hemorrhage of the purpuric type. Although Pollak and Riehl are unable to disregard absolutely the possible signficance of a toxic effect on the vasomotor apparatus of the blood vessels in these cases, they believe that this is a factor only in a certain number of cases. It is obvious that the vascular spasms and angioneurotic edema observed in other organs following the administration of arsphenamine may occur also in the brain, so that constriction of the arterioles may lead to a slowing of the circulation in the capillaries and to stasis with diapedesis and secondary necrosis of the surrounding tissues. The authors, however, believe, as do Fischer and Tannenberg as well as Razek, that even in these cases one is dealing with a disease of the endothelium and that the circulatory changes are due chiefly to this involvement. Under this type is also included a group of cases in which, as a result of vascular involvement, there occurs a nutritional disturbance of the nerve tissues with consequent softening. These cases are usually combined with hemorrhage. Isolated softenings within hemorrhagic areas are rare in the authors' experience.

2. The lesion in this type is a primary involvement of the neuroglia in the presence of an apparently normal circulation and an intact vessel wall. The neuroglial involvement may be associated with pathologic changes in the adjacent nerve parenchyma. The pathologic picture may resemble that of a white cerebral softening in which there is no definite evidence that the lesion is due to vascular changes, although in recent years such a process has been attributed in numerous instances to vasomotor causes. The authors believe that in these cases the toxic effects of the arsphenamine produce an insufficiency of the neuroglia. Depending on the extent and intensity of the neuroglial involvement, further sequelae may arise; of these the most important are processes of softening of the nerve parenchyma with edema or swelling due to circulatory disturbances in the cerebral fluid spaces. This disease of the neuroglia is, in the final analysis, analogous to involvement of the endothelial layer of the blood vessels as in type 1, because from a functional point of view the neuroglia resembles to a certain extent the endothelial system; like the latter, it aids in the resorption of nutritive substances and in the permeability of the various body juices.

3. This type is a combination of types 1 and 2. The sharp limitation between types 1 and 2 may in some cases be extremely difficult.

4. This type includes the cases in which there occurs a productive Herxheimer reaction in the central nervous system. (Under this heading the authors include only cases with definite morphologic evidences of a specific productive reaction in the tissues; otherwise every case of arsphenamine intoxication could be designated as a Herxheimer reaction. Their conception of a Herxheimer reaction is an arsphenamine toxicosis which seems to affect primarily the spirochetes, as a result of which endotoxins are produced that give rise to a secondary productive reaction in the tissues. Whereas the predominating histologic process in the other three types is either in the vascular endothelium or in the neuroglia, or in both, in the fourth type the predominating pathologic process seems to be one of a productive defensive process of the mesodermal tissues with an active hyperplasia of ectodermal glial tissue. In this sense one must look on the energetic development of lymphocytes, plasma cells, endothelial and adventitial cells, as well as on the extensive and intense perivascular and pericellular glial proliferation, as

evidence of a tissue defense against spirillar toxicosis. In a morphologic sense this process may be regarded as similar to that of the production of an antitoxin. It is obvious that in a severe case of this type there may be found in the center of the process evidences of regressive changes and even necroses—a possibility that may be encountered in every hyperplastic process—but this does not invalidate the theory that the process represents essentially a secondary reaction. Of course, there is no reason why the spirillotoxic effect of arsphenamine cannot be combined with its "tissue toxic" effect, as in types 1 and 2, and give rise to combined lesions in the same case, although the authors state that in their material they have never observed such a combination of lesions following arsphenamine intoxication.

The problem becomes more complicated when one also takes into consideration the labile effect of arsphenamine on the vasomotor system; the changes in vascular tonus thus induced must also have some effect on vessel permeability first, and later on the proliferating and regressive changes in the mesodermal elements.

Arsphenamine, therefore, may exert its toxic effects on different tissues in different cases. The question then arises, why does not the Herxheimer reaction occur in every case? The authors answer: It is well known that in all cases in which a Herxheimer reaction occurs in the central nervous system, the latter shows no clinical evidences of involvement. It would seem, then, that in these cases the spirochetes exert a sudden effect on the nerve tissues and on the vascular apparatus with an entirely different result from that observed in cases with involvement of the central nervous system in which the spirochetes have exerted their deleterious effects for a number of years, and thus mobilized slowly and gradually the various reactions in the vessel walls, neuroglia and brain tissue. This slow and gradual process permits these tissues to adapt themselves gradually to the chronic effects of the syphilitic poison with a fairly continuous defensive process against the sudden invasion of the spirochete and its toxin. Under certain conditions these factors do not exist in the second stage of syphilis, so that a fresh invasion of spirochetes into the cerebral vessels, damaged by the administration of arsphenamine, produces an acute syphilitic disease of the mesoderm with secondary involvement of the neuroglia. Such typical reactions would appear much more often were it not for the fact that the arsphenamine injures precisely those tissues that are essential for the development of the typical productive components of the Herxheimer reaction. A typical degenerative disease of the vessel wall and of the neuroglia prevents the development of a productive Herxheimer reaction, because a breaking down of these tissues makes it impossible for the appearance of the productive reaction, and hyperplasia never occurs in a degenerative process. This, the authors believe, may possibly be the reason why type 4 of the arsphenamine reaction is observed so rarely in practice. At all events, it would seem that all injuries following the administration of arsphenamine are eventually due to the toxic effects of the latter, and the nature of the histologic process depends on the lack of resistance of the tissues to arsphenamine.

The next question that presents itself is: What factors determine the predominance of the type of lesion and its localization following the administration of arsphenamine? One of the factors in the cases in which the blood vessels are predominatingly involved may perhaps be an associated disturbance of renal function, owing to which a greater burden is cast on the vessels, so that these are more readily damaged by the arsphenamine. In other cases it may be that under certain circumstances there occurs an increase in the water content of the cerebral tissues through which the neuroglia is considerably overburdened, so that they are more readily affected by the toxic effects of arsphenamine with a resulting glial necrosis. In still other cases one may be dealing with a latent infectious disease in the course of which both the blood vessels and glia have been so damaged that they succumb readily to the toxic effects of the arsphenamine. The authors admit that all reasons advanced by them are purely theoretical and still remain to be proved pathologically; this, however, they state cannot be expected until more definite knowledge has been acquired as to the toxic effects of arsphenamine on living tissue.

KESCHNER, New York.

A Case of Disturbance of Associated Movements of the Eyes, with Nystagmus Retractorius and Dissociation of the Vestibular Nystagmus. J. Nordmann and O. Metzger, Rev. d'oto-neuro-opht. **8**:17 (Jan.) 1930.

An unmarried woman, aged 23, entered the clinic on Jan. 4, 1930, complaining of troubled vision. She felt well and had never been sick and her family was in good health. Two weeks previously, she suddenly had the sensation of rotation in the head. A complete examination revealed no abnormalities worth mentioning, except an inconstant deviation of the outstretched arms, a suggestion of falling to the left in the Romberg test and the following difficulties in the extrinsic muscles of the eyes: on looking straight ahead, the ocular globes were parallel, with at times a few jerking movements; on convergence, these became more regular, quick and of small amplitude and were in the vertical plane. When the patient was directed to look downward, the movements were normal; on looking to the left and to the right, there was a horizontal nystagmus, which in the latter case was accompanied at times by retraction of the right globe; this retraction was still clearer when the patient attempted to look upward. The eyes could not be raised upward above a position 10 degrees above the horizontal; when this position was obtained, there was a feeling of malaise and a sudden marked convergence, and the globes were retracted in the orbit. A slight crossed diplopia was found, the image of the right eye being always above that of the left. These disturbed movements were noted in the guided movements of the eyes as well as in the command movement, but were lacking when the vision was fixed and the head lowered. With one eye covered, the same phenomena of retraction and convergence on looking upward and nystagmus in lateral movements were noted on both sides, but on looking to the right, retraction of the globe was present only on examining the right eye. Optokinetic nystagmus was absent in the vertical descending direction. On douching the left ear with cold water horizontal nystagmus to the right was produced in the left eye and a vertical upward nystagmus in the right eye; on inclining the head toward the right shoulder, the horizontal nystagmus in the left eye became rotary, and the vertical nystagmus in the right eye was amplified and slowed; when the head was inclined forward 90 degrees, the horizontal nystagmus in the left eye was inverted, and there were deviation of the arms and body to the left and slight vertigo. On douching the right ear, a horizontal nystagmus to the left of good amplitude was produced in the right eye and a vertical upward nystagmus in the left eye; with the head on the right shoulder, the nystagmus became rotary; with the head forward 90 degrees the nystagmus became horizontal to the right. After rotation to the right, the nystagmus was horizontal to the left in both eyes; after turning to the left, horizontal nystagmus was produced in both eyes, but it became vertical in the right eye as soon as its intensity diminished.

The disturbed ocular motility remained the same for a week and then it improved rapidly; commanded movements became normal and retraction of the globe disappeared, but convergence and looking upward were still accompanied by vertical nystagmus. The caloric test still produced perverted nystagmus, different in the two eyes. At the last consultation on January 30, the ocular motility was normal, but the spontaneous vertical nystagmus persisted. The caloric test gave normal and equal ocular reactions.

The interesting points are: 1. The nystagmus retractorius is very rare, only seven cases being found in the literature. The retraction may be produced by the simultaneous contraction of all of the ocular muscles, and the recti being stronger than the oblique muscles, a retraction results. It appears, however, that simultaneous innervation of the four lateral recti is sufficient to produce this curious result. 2. The dissociation between the commanded movements and the automatic reflex movements excludes a lesion of the nuclei and the peripheral oculomotor apparatus. In the case reported, the lesion was supranuclear. 3. The optokinetic nystagmus was normal upward and was abolished downward. For the production of the quick component downward, there must first be a slow move-

ment upward, which was lacking in this patient. The conclusion is that the slow component of optokinetic nystagmus is only a guided associated movement in relation with fixation. 4. Vestibular disturbances were represented by the spontaneous nystagmus and, after the caloric stimulation of one labyrinth, by a dissociated nystagmus. In order to explain the dissociation, a disturbance of the crossed vestibulo-ocular fibers must be assumed, a disturbance which enfeebled the stimulus from the labyrinth of the opposite side, permitting the spontaneous vertical nystagmus to persist. The rotation test confirms this view, but the inversion of the horizontal nystagmus speaks against such hypothesis.

Nystagmus retractorius, in the known cases, has always been caused by diffuse peduncular lesions. Salus found a hydatid cyst of the sylvian aqueduct, Elschnig, a tumor of the third ventricle and aqueduct and Bárány, a tumor of the quadrigeminus. The symptoms in the case reported speak for a peduncular lesion, situated at a point where the superior vestibular and the supranuclear oculomotor pathways are close together. A peduncular lesion must, besides, leave the nuclei of ocular motility intact. Owing to the rapid evolution of the phenomena and to the absence of previous illnesses, the authors believe the cause to be an encephalitic or toxic lesion rather than a beginning manifestation of multiple sclerosis.

DENNIS, Colorado Springs, Colo.

TRUTH, ERROR AND FALSEHOOD—HUMAN AND BIOLOGIC. C. VON MONAKOW, Schweiz. Arch. f. Neurol. u. Psychiat. **25**:207, 1930.

Von Monakow believes that the psychologists should not only approach their problems from a biologic or objective point of view, but that they should also study the parallel development of function and structure in the central nervous system, never forgetting the time element. Introspection should serve merely to throw light on affective phenomena.

Truth, although of interest mainly to philosophers, psychologists and jurists, could well be studied by neurologists from the point of view of the development of mental function, the organization of the instinctive world and the development of the central nervous system. From a biologic point of view, truth may imply either a faithful reproduction of one's own personal ideas and feelings (interoceptive truth), or a true reproduction of external conditions and relations as they appear to us through the medium of our sense organs and our trained and logical mental processes (exteroceptive truth). Only the latter variety is capable of verification by others with approximately similar mental instruments.

The author discusses a development of mental functions which he believes are located mainly in the cortex, under the influence of the individual's instinctive trends, his immediate and future needs and his environmental relations, emphasizing the individual differences which must exist. He thinks that logical thought must develop as do skilled motor acts, in which the character of each step must effect the entire process.

An individual's observations of a series of events are always more or less incomplete, and gaps are apt to be filled in accordance with instinctive striving and unconscious desires, as is attested by the experiences of judges with witnesses. In the physical sciences, truth seems to rest on the most solid foundations, based as it is on accurate measurements of phenomena that can be confirmed by others, but even here generally accepted laws may be found eventually to be only approximations, and differences of opinion may exist between equally well trained observers. In the less exact biologic sciences one is compelled to deal with probabilities and approximations.

Common forms of expression are inaccurate in many instances. The author decries especially the lack of proper terms for affective phenomena. Descriptions of character leave much to be desired. What is said of an individual may be true only at a certain time and under certain conditions. Actions generally considered to be reprehensible spring from instinctive tendencies, present in all of us to some degree.

In his early development the individual must separate the essential from the unessential in his sensory impressions, and this selection is controlled mainly by the instincts and the desires of the moment. Much that may later become important is disregarded. While constant corrections occur with increasing experience, many early formed habits of thought persist.

The individual is especially apt to make unintentional false statements (errors) under the influence of secret hopes and fears in describing external conditions in which his personal interests are involved. He may also mis-state his own private feelings or beliefs with no intention of lying for the same reason. When he becomes conscious of the error, he may experience feelings of discomfort (Unlustgefühle).

A lie is an intentional falsification as a means of self-defense, escape from a situation or of acquiring some immediate advantage. "Biologically considered, the lie is a functional wrong way in organized protoplasm, a temporary betrayal of the personal and superpersonal future interests in favor of a momentary satisfaction of a personal desire, a satisfaction which from its nature can only be short." After attainment of the desired results, there is usually a reaction in the interest of the future welfare of the protoplasm with feelings of shame and disturbances in the internal secretory and autonomic nervous systems. Through overcompensation the reaction may become diminished in intensity without being entirely subdued. Habitual lying may produce a neurosis.

In conclusion, the author decides that strictly speaking every truth is only human and relative. In the cosmos, however, there are only self-evident facts (Selbstverständlichkeiten).

DANIELS, Rochester, Minn.

CEREBROSPINAL AND OCULAR FLUIDS IN EXPERIMENTAL JAUNDICE IN DOGS. HARRY L. SEGAL and JEROME GLASER, Arch. Path. **9**:1038 (May) 1930.

Recent studies of the cerebrospinal fluid of infants, indicating the frequency of xanthochromia, have stimulated further investigation of the relationship of xanthochromia in the spinal fluid to clinical jaundice. The literature relating to the subject of icterus and xanthochromia in the spinal fluid has been reviewed by Glaser; from it, conflicting evidence is noted. Some investigators assert that the bile pigments are present in all cases in the spinal fluid in jaundice. Others have found it in only a few cases; still others only in long-standing and severe jaundice. The icteric cases studied were usually of the obstructive type of jaundice with varying etiology. The ocular fluids were not mentioned in these papers.

In the work here reported there was a slight yellow tinge to the spinal fluid in five of the eleven adult dogs. The benzidine test for hemoglobin and the van den Bergh reaction for bile pigment were negative in every instance. The lowest icterus index of the blood serum in which there was an accompanying yellow cerebrospinal fluid was 150. The icterus index of the blood serum ranged as high as 182 without xanthochromia of the spinal fluid. This, perhaps, suggests a threshold for the appearance of xanthochromia under the conditions of the experiments. This is particularly probable since, with icterus indexes as high as this, owing to the difficulty of exactly matching yellow colors, a difference of 30 points may be within the limits of experimental error. The same general facts hold true for the aqueous humor, but with this fluid the xanthochromia became more intense than in the case of the spinal fluid and the van den Bergh reactions were positive. The vitreous humor remained colorless in all experiments. The yellow aqueous humor occurring in these experiments suggests a possible explanation of xanthopsia in some cases of jaundice.

The authors were unable to produce any marked icterus in young dogs comparable with that in adult dogs. Toluylendiamine seemed to produce a severe anemia in the former without producing an intense jaundice. In one case, in which the icterus index of the blood serum reached 28, there was a definite yellow tinge to both the spinal fluid and the aqueous humors, but the van den Bergh reactions in both fluids were negative.

The xanthochromia occurring in the spinal fluid of dogs in the presence of negative van den Bergh reactions is probably due to the presence of bile pigment in insufficient amounts to give such a reaction, but sufficient to produce a faint yellow color. To determine whether this might be possible, the authors diluted some icteric dog serum with physiologic solution of sodium chloride until a point was reached at which a faint yellow color persisted, but the van den Bergh reaction was negative. The concentration of bilirubin in this solution was 1:1,650,000, if one considers that an icterus index of 100 roughly matches 100 mg. of pure bilirubin per liter. This dilution is thus just beyond the 1:1,500,000 dilution of pure bilirubin in which a positive van den Bergh reaction is still obtained.

In the experiments reported, the icterus index of the xanthochromic spinal fluids never exceeded 1; the icterus index of the yellow aqueous humors ranged as high as 4. The vitreous humor always remained colorless. The van den Bergh reactions were always negative in the xanthochromatic spinal fluids, but positive in the aqueous humors which had an icterus index of 3 or 4.

WINKELMAN, Philadelphia.

THE TEMPERATURES IN THE TISSUES WHICH ACCOMPANY TEMPERATURE SENSATIONS. H. C. BAZETT, B. MCGLONE and R. J. BROCKLEHURST, J. Physiol. **69**:88 (March) 1930.

The authors recorded photographically measurements of temperatures within the tissues coincident with temperature sensations. Temperatures were recorded by thermocouples introduced into the skin of the forearm in the immediate area of the warm and cold spots. The stimulus used was an esthesiometer of the Thunberg type, either 1.5 or 11 mm. in diameter. During the period of preparation the bare forearm was exposed to the room conditions for at least one hour, and after the introduction of the thermocouples thirty minutes elapsed before recording, in which time any inflammatory reaction was subsiding.

A series of stimulations of the end-organs was made at intervals which were only occasionally less than five minutes. Most of the experiments were without experimental variation of the circulation. A few experiments were repeated after an inflammatory reaction had been produced in the surrounding area.

Data are presented illustrating the magnitude of the temperature changes on the surface and within the dermis, with various stimuli and with applicators of different size. The magnitude of the temperature change is demonstrated to be a function of the size of the applicator and the strength of the stimulus. The skin during stimulation does not attain the temperature of the applicator as usually has been assumed. The main thermal change penetrates the tissue with a velocity of 0.5 to 1.0 mm. per second and the rate varies with the vascular conditions.

Vascular dilatation shows the rate of penetration (with the possible exception of cold in the most superficial layers). Warmth is conducted more rapidly than cold under all conditions except in the superficial layers of the skin. Latencies of sensation were measured under various environmental conditions and showed variations according to the vascular state.

Latencies for both cold and warmth were longer in a warm room. Inflammation interfered with sensations of cold and exaggerated their latency; it also interfered with paradoxic sensations of cold. By correlation of latencies of sensation with the rate of penetration, estimates of the depth of the end-organs concerned were made. These are 0.15 ± 0.1 mm. for the end-organ for cold, and 0.6 ± 0.2 mm. for warmth. The end-organ for warmth cannot lie in the subdermal tissue (i. e., deeper than from 1.5 to 2.0 mm. in the forearm). Paradoxic sensations of warmth were noted in response to cool stimuli and appeared facilitated by inflammation.

Description is given of complicated curves which may be recorded at the greater depths and which sometimes have indicated temperature changes opposite in sign to those anticipated from the stimulus. These changes appear to be due to variations in blood distribution produced by the pressure of the applicator, and

are not related to paradoxic sensations. After-sensations of both cold and warmth were recorded; their relation to changes of temperature was discussed. Bazett and his co-workers believe that the data do not warrant discussion of the mode of stimulation of the end-organs at present; more exact information of the anatomic structures concerned is necessary.

ALPERS, Philadelphia.

SIZE OF OBJECTS IN RELATION TO THEIR VISIBILITY AND TO THE RATING OF VISION. C. E. FERREE and G. RAND, Arch. Ophth. **4**:37 (July) 1930.

In this extensive article the authors discuss three important factors in the visibility of objects, i. e., the size of the visual angle of the detail to be discriminated, the difference in the coefficient of reflection between the object and the background and the intensity of the illumination. They describe the methods used and give in detail the calculations from charted results. The charts, curves and recapitulations are all included.

Ferree and Rand find that the size of the stimulus is the most important of the three factors mentioned. Further, the size of the object and the difference in the coefficient of reflection between the object and the background influence the speed of vision in several ways, which are given. The speed of vision—taken as the reciprocal of the smallest interval of time in which the discrimination of detail can be made, the time threshold of discrimination—is a sensitive, feasible and important measure of the relation of the size to the visibility of objects. The observations demonstrate: (1) the relationships between the speed of vision and the degrees of illumination by increasing the size of the object; (2) the disproportionate ratio of the increment of speed per unit increment of the visual angle as compared to the increase in the visual angle; (3) the speed of vision when studied for white objects on black, black objects on white, white objects on gray and black objects on gray, and (4) the effect that the difference in the coefficient of reflection between the background and the object plays on the speed of vision. Furthermore, visibility as measured by the speed of vision varies greatly with the intensity of the illumination and the relation of the object to the background, and is in general greater than the visibilities rated in terms of the visual angle. Absolute visibility increases with an increase of the intensity of illumination for all sizes of objects and all relations of the object to the background; but a relative visibility exists in which the deviations of ratings as measured by speed from those made in terms of the visual angle become smaller as the intensity of illumination is increased. The differences for both absolute and relative visibility are given which exist for all sizes of test objects and intensities of illumination when comparing black on white, and black on gray and, under similar conditions, white on black, and white on gray. The industrial relationships of the speed of discrimination, the intensities of light, the size of objects and the differences between the object and the background are also discussed. The authors give the difference in rating between the visual angle and the scales of speed for the size of objects, the intensities of illumination and the difference between the object and the background.

Other conclusions as to allied factors that modify different ratings of visibility, efficiency of work and the use of the eyes complete a most valuable work in optics.

SPAETH, Philadelphia.

EFFECT OF STRAMONIUM IN PARKINSONISM. EDWARD D. HOEDEMAKER and M. A. BURNS, J. A. M. A. **95**:91 (July 12) 1930.

Dr. Stoerck introduced *Datura stramonium* in the cure for epilepsy, mania and some other convulsive conditions. In 1798, Cooper employed the drug for the relief of mania, epilepsy and certain fevers. Since then it has been widely used for the aforementioned ailments as well as for headache, insomnia and muscular rheumatism. Recently, stramonium has been employed in the treatment for parkinsonism, especially of the postencephalitic type. A brief review of the

literature follows. There is then a paragraph on the composition of and pharmacology of stramonium.

The authors report the effect of tincture of stramonium in a series of thirty-one cases. Four were classic paralysis agitans and twenty-seven were the syndrome following epidemic encephalitis. A 10 per cent tincture was employed and given in doses of 20 minims (1.25 cc.) three times daily, each dose being increased 5 minims (0.3 cc.) a week until a dosage of 60 minims (3.6 cc.) or more three times a day was reached. The four patients with paralysis agitans experienced but little improvement. There were nine postencephalitic cases in women—aged from 20 to 43—and eighteen in men—aged from 20 to 70. There was less response in the more severe cases.

In eleven cases there were excellent results, such as marked diminution in rigidity, a surprising return of associated movements and marked improvement in the general mental condition as well as complete relief from excessive salivation. In five cases there was not quite such complete relief, and in five only fair results were obtained, while there were six in which relief was experienced only from excessive salivation.

Under therapeutic effects the authors consider rigidity, tremor, excessive salivation, automatic associated movements, oculogyric crises and mental effect. The average dose was 76 minims, (4.56 cc.), three times daily. Younger patients showed greater tolerance, and men showed greater tolerance for the drug than did women. The toxic effects noted were xerostomia, blurring of vision, dizziness, loss of appetite, nervousness, and insomnia and pruritus. Nausea and vomiting did not occur.

In summary, in the twenty-seven postencephalitic cases, three patients have been discharged and have returned to work, ten others are in a condition to be discharged and the rest are still under observation.

Hoedemaker and Burns conclude that: (1) tincture of stramonium lessens the rigidity in parkinsonism and increases the ability to perform fine, rapid movements; (2) tremor is rarely noticeably affected; (3) the mental condition is improved; (4) the maximum dosage varies from 60 to 90 minims (3.6 to 5.4 cc.) three times daily; (5) toxic effects are not severe; (6) cases of classic paralysis agitans are little benefited by stramonium, and (7) tincture of stramonium is a drug of value for patients afflicted with parkinsonism and should be more widely used.

CHAMBERS, Syracuse, N. Y.

HISTORY: CONTRIBUTION TO HISTORY OF THE THEORIES OF LANGUAGE IN NORMAL AND PATHOLOGIC STATES. THE IDEAS OF EDWARD FOURNIÉ (1866 AND 1872). R. MOURGUE, Schweiz. Arch. f. Neurol. u. Psychiat. **25**:245, 1930.

The works published by Fournié in 1866 and 1872, although not mentioned by well known writers on aphasia, reveal views on the nature of language that were far in advance of the author's time. He not only had good philosophical and scientific training, but he worked for many years as an otologist in the National Institution for the Deafmutes, at Paris. Consequently, he was in a better position to study the physiology of speech than were his better known contemporaries. He criticized existing theories and formulated a theory of his own which he incidentally applied to the question of aphasia. Fournié believed that the function of language could not be studied apart from the entire thinking process. He believed that ideas were not merely sensations, but were the sensations transformed by the intelligence into voluntary movements, the voluntary movements constituting the elements of language. Speech being an essential part of thinking, the mind could not develop beyond a primitive level without this function. Written speech merely represents visual translation of that spoken, for, as he said, we speak mentally as we read. He also recognized the importance of muscle sense and hearing in the control of speech. In direct contradistinction to the accepted view of his time, he thought that internal speech was necessarily secondary to external speech.

The existence of a regulatory center for speech was denied by this author because such a center should include all the elements which go to produce speech, and that would be impossible as these elements are scattered throughout the cortex. In regard to aphasia he supposed that in Broca's area were located peripheral cells representing auditory perceptions in the state of notions and that from these cells impulses pass to the striate body, which he supposed controlled voluntary movement. This did not mean, however, that the function of speech was located in Broca's area. He thought that aphasia could also be caused by a lesion of the parts that regulate speech, namely, the auditory nerve or its connections with the centers receiving from the intelligence the impulse necessary to execute the voluntary act.

Mourgue, the author of the paper, believes that the writings of Fournié are to be compared to those of Hughlings Jackson, for although not possessing the depth of the latter, they seem to anticipate to at least some degree modern views on aphasia.

GUSTATORY FACIAL HYDRORRHEA. PAUL REBIERRE, Rev. d'oto-neuro-opht. **8**: 23 (Jan.) 1930.

A man, aged 37, was operated on in 1921 for chronic cervical adenitis of the right side, leaving a cicatrix, 6 cm. long, which began over the parotid area and extended down along the border of the sternocleidomastoid. Sensations of smothering, present before the operation, increased after it. During the crises, he had a feeling of an arrest of respiration, an inability to move, anxiety and pain extending from the cicatrix to the parietal region and periphery of the ear. The attacks came on while alone, but often, especially in public, while chewing and swallowing certain foods. Profuse sweating occurred, localized strictly to the right side of the face from the hair line down to a line running horizontally from the external auditory meatus to a point midway between the nares and the eyelid. The attacks sometimes came on between meals when intestinal digestion was difficult. The attack was followed by fatigue and inability to fix the attention. Palpation of the cicatrix produced pain similar to that produced spontaneously, a redness of the conjunctiva and tears. The voluntary motility of the face was good, but on smiling only the left half of the face was active.

An examination of the eyes revealed a normal condition, although cocaine and homatropine caused only slight dilatation of the pupils. Other examinations, including the Wassermann test, gave negative results.

The affected zone did not correspond to the territory of distribution of any cerebrospinal nerve. Probably the sympathetic system was at fault and the theory of André Thomas—after lesions of the nerve the regenerated fibers are switched and the reflex pathways are changed—applies. In wounds of the sympathetic system, it is not always a gustatory stimulus that causes a localized sudorific reflex. The stimulus may come from a nearby or a remote irritation of the sympathetic system. A prerequisite, however, is a state of hyperexcitability of the vegetative system, either preexisting or caused by the lesion itself.

There existed in the reported case evidences of vegetative paralysis. In the depths of the tissues, nerve fibers were destroyed or compressed by the scar or irritated by neuromas. It is to be noted that the particular food which excited the reflex is one that did not agree with the patient, and that the reflex was also caused by defective digestion. Thus, the phenomenon was a sort of defense reflex.

DENNIS, Colorado Springs, Colo.

EARLY PSYCHIC INVALIDISM. J. KASANIN, New England J. Med. **202**:942 (May 15) 1930.

The author describes briefly the disease entity which was first formulated by Gannushkin under the name of "acquired psychic invalidism." This is a reaction type which has apparently evolved out of the peculiar conditions that have followed the revolution in Russia. It is usually found in comparatively young persons

between the ages of 20 and 30 years. Clinically, it shows a group of symptoms some of which are not unlike those found in the exhaustion states of persons of neurotic make-up. To these are added marked intellectual impairment and disturbances of emotional tone. The latter do not seem to resemble any of the secondary symptoms of the known functional diseases, but approach most nearly the reactions found in the early stages of cerebral arteriosclerosis. In most cases the history is that of unusual mental exertion, frequently accompanied by actual physical factors such as concussions, infectious diseases (typhus and typhoid) and toxic factors such as alcohol and cocaine.

A typical case is reported in which the disease developed following pronounced exertions in attempting to carry a position for which the patient had no adequate training, a great many physical exhaustion factors, a definite concussion and a prolonged attack of typhoid fever.

In a group of these cases Steffko undertook histologic studies and found a number of organic lesions, such as adhesions between the meninges and the brain, hemorrhages, hyaline degeneration of the vessels, sclerosis and shrinking of the ganglion cells and in some cases edema and foci of softening. According to the opinion of both of these observers, this symptom complex should be regarded as a definite disease entity in the nature of a premature aging of the brain tissue. They believe that the rôle played by the infections, toxemias or concussions is essentially a secondary one, because the central nervous system of these persons has been rendered more vulnerable to such factors. With the knowledge that has recently been brought to light of the possible organically conditioned mental states that can actually follow such physical factors, it is somewhat doubtful whether one would be justified in speaking of a special disease entity in cases in which so many different etiologic possibilities have to be taken into consideration.

MALAMUD, Iowa City.

CYCLIC OR RHYTHMIC OCULOMOTOR PARALYSIS. E. SELINGER, Arch. Ophth. **4**: 32 (July) 1930.

This article is a report of the twenty-ninth recorded case of a rare and interesting oculomotor condition. The author states that it is the first to be presented in American literature. The symptomatology in his case is given in detail. The patient had had two or three operations in early childhood for squint; unfortunately the data for these was unobtainable. The left eye was normal.

The Wassermann reaction of the patient's blood was negative, and that of the spinal fluid, positive. The oculomotor palsy presented a cyclic syndrome of two phases: a primary flaccid stage of about twenty seconds' duration with ptosis of the upper lid, divergent strabismus and a pupil dilated to 8 mm.; a secondary spastic phase of from fifteen to twenty seconds' duration during which the outer half of the eyebrow moved upward with wrinkling of the skin of the forehead, the lid was elevated in a jerking manner until the palpebral fissure was from 8 to 9 mm. in width, the internal rectus contracted spasmodically, diminishing the divergent squint from 5 to 8 degrees and the pupil contracted to about 3 mm. The entire cycle occupied from sixty to eighty seconds and was repeated constantly. Spasmodic contraction of the ciliary body accompanied the spastic contraction of the pupil. Light, convergence and accommodation reflexes reacted as one would expect during the flaccid as well as the spastic phases. Other signs of cyclic spasticity were consistent. The fields and fundi had no signs of a pathologic process.

The review of the literature is apparently complete. It covered the many ocular motor forms which have presented themselves and the multiplicity of the symptoms, and it included a discussion as to the probable microscopic process that accounted for these symptoms. The theories advanced are common only in that the paralytic phase is undoubtedly the primary one, the spastic being secondary or subsequent. There seems to be, as well, some relationship to syphilis, especially in regard to the treatment.

SPAETH, Philadelphia.

OBSERVATIONS OF A FEW MYOTONIC CONDITIONS. GEORG PAMBOUKIS, Schweiz. Arch. f. Neurol. u. Psychiat. **26**:84, 1930.

In this paper, which was presented before the Medical Society in Athens, the author relates some interesting experiences in cases of myotonia. The first case was one of typical Thomsen's disease in a man whose mother had suffered from diabetes insipidus and who had been relieved by a solution of pituitary. After forty injections of solution of pituitary, this man regained almost complete freedom of movement. In the second patient, a man, aged 68, myotonic symptoms developed in the hands, forearms and legs following a period of overexertion and exposure. The author demonstrated the typical myotonic reactions to electric and mechanical stimuli and succeeded in permanently relieving the patient of his difficulties with calcium chloride and acetylsalicylic acid. The third patient, a busy surgeon, had noted myotonic symptoms in the hands for ten years. Following an attack of dengue fever, the patient experienced difficulty in opening the closed eyelids, and the author was able to demonstrate myotonic signs in the muscles of the hand, forearms, eyelids and tongue, and to a lesser degree in other muscles. Temporary but not permanent relief was obtained by calcium chloride and acetylsalicylic acid, the latter medicine appearing to be the most effective. The fourth patient, a woman, aged 28, had ceased menstruating at 34 years. Subsequent to an attack of dengue fever she lost most of her hair and experienced difficulty in opening her eyes. Myotonic symptoms were pronounced in the orbicularis palpebrarum and existed to a lesser extent in other muscles. The blood calcium was somewhat reduced (0.008 per hundred cubic centimeters). Considerable relief was afforded the patient by treatment with an ovarian preparation, calcium chloride and acetylsalicylic acid.

The author believes that myotonia is to be explained on the basis of humoral and parasympathetic action on the sarcoplasm. The toxin of dengue fever is believed to stimulate the parasympathetics, and in the fourth case reported this disease seemed to bear a direct causal relation to myotonia. In this case pilocarpine increased the myotonia while epinephrine relieved it. The author fails to mention that the sympathetics rather than the parasympathetics are generally considered to control the sarcoplasm in the hypothetic rôle which it plays in the production of muscle tone.

DANIELS, Rochester, Minn.

REFLEX SUMMATION IN THE IPSILATERAL SPINAL FLEXION REFLEX. J. C. ECCLES and C. S. SHERRINGTON, J. Physiol. **69**:1 (March) 1930.

The central condition produced by a single volley of afferent impulses in an ipsolateral flexor center has been investigated by Eccles and Sherrington by sampling with a second afferent volley (usually in another nerve) at various intervals of time after the first. Under these conditions, when each reflex stimulus was just threshold, reflex responses were produced at certain stimulus intervals. This can be explained only by the effects of central summation. Curves can be drawn in which the intervals between the stimuli are plotted against the tensions of the reflex responses. With the twin nerves of the gastrocnemius acting as afferents reflexly on the tibialis anticus, there is often an optimum degree of facilitation at simultaneity of the stimuli, and also with the stimulus to one or the other of the nerves, leading by an interval of about 12 T. This double effect was traced to two separate central excitatory states, one "direct," the other "delayed." Evidence is given that this second effect is evoked by the tension receptors of the gastrocnemius, and is possibly a rebound following an initial inhibition. There is thus a complex central condition following a single afferent volley. When the semitendinosus was similarly excited from various pairs of nerves, complex curves were also obtained.

The authors believe that the most striking feature of the results has been the comparatively long duration of the central effect of one threshold stimulus as shown by its power of facilitating the effect of a second threshold stimulus. This shows that the first single afferent volley must produce an enduring central state — the

central excitatory state—which is capable of summation. Moreover, since it is found that facilitation between threshold stimuli is almost universally demonstrable, it would seem that a single afferent impulse is by itself unable to produce a threshold central excitatory state in a neurone. Summation is necessary for every reflex discharge. Reflex threshold or neuron threshold is to be distinguished from afferent nerve threshold. The enduring central excitatory state is responsible for after-discharge, an extreme example of which is described in a deafferented tibialis anticus of a dog with a chronic lesion of the spinal cord.

ALPERS, Philadelphia.

HEADACHE IN OPHTHALMIC MIGRAINE. CANTILO, Rev. d'oto-neuro-opht. **7**:639 (Oct.) 1929.

Ophthalmic migraine comprises three phases: (*a*) the premonitory signs, (*b*) the visual crises and (*c*) the algic phase and its accompanying disturbances. A severe orbital pain persisting for several hours precedes the typical visual crises. This pain is of dural origin, in the occipital region near the cuneus, the point where the phenomenon of vasoconstriction is produced. A case published by Hedon and Bosquet illustrates this. It concerned a patient who had been operated on for lateral sinus thrombophlebitis. Irritation of the inflamed dura at the posterior angle of the wound caused a violent pain in the orbit, lasting several hours. This phenomenon was explained by the fact that the innervation of the tentorium occurs by way of the recurrent nerve of Arnold, a branch of the ophthalmic; irritation of the terminations of this branch caused pain in the sensory territory of the ophthalmic. The recurrent nerve of Arnold is likewise irritated in ophthalmic migraine by an intense vascular disturbance in the dura surrounding the visual centers.

Didsbury has established the presence of fixed painful points along the course of two important branches of the trigeminus: the external frontal and Arnold's nerve. These painful points are caused by a chronic superficial perineuritis. They are brought out by light pressure. The presence of this sign, located above the median half of the brow, is a valuable diagnostic observation.

True ophthalmic migraine, in contradistinction to the innumerable ophthalmic pseudomigraines, is accompanied by characteristic pains: pains during the crises and, between the crises, fixed painful points, particularly arising from the external frontal nerve. The importance of this sign in differentiating ophthalmic migraine from migraine accompanying ophthalmoplegia is great. The prognosis in the former is benign and in the latter grave. The orbital pain in the former almost always follows the visual disturbances and appears only exceptionally between the crises.

DENNIS, Colorado Springs, Colo.

STUDIES OF THE PULSE AND BLOOD PRESSURE IN CATATONIC PATIENTS IN A NORMAL STATE AND AFTER THE ADMINISTRATION OF DRUGS. F. WALTHER, Arch. f. Psychiat. **89**:377, 1930.

The author reports investigations of the pulse and blood pressure and their relationship to one another in normal as well as in catatonic persons. These studies are supplemented by investigations of the same type in the same persons after the administration of different drugs. Four normal persons were studied and the conclusions reached were that a pulse of 60 corresponds to a systolic pressure of from 115 to 135 and diastolic of from 60 to 80 with a pulse pressure of from 50 to 70. A pulse of 70 normally corresponds to a systolic blood pressure of from 120 to 145, a diastolic pressure of from 60 to 90 and a pulse pressure of from 50 to 80. Drugs that cause an increased irritability in the sympathetic system will bring about an increase of the pulse rate and of the systolic pressure with a decrease of the diastolic pressure, thus causing an increased pulse pressure. On the other hand, an increased irritability of the parasympathetic will cause a decrease both in pulse rate and in pulse pressure. Atropine first causes stimulation of the

vagus but later paralysis. Pilocarpine is a sympathetic stimulant. Epinephrine acts mostly in a similar fashion. Pituitary extract is a vagus stimulant and testicular extract acts in the same way; so does thyroxin to a certain extent, although the author states that other observers found this drug to have the opposite effect. The effects of drugs in a normal person depend much on the type of personality. In a sympatheticotonic person, drugs that stimulate the sympathetic system will have more effect than those that stimulate the vagus, and vice versa.

Catatonic patients (five cases were studied) differ from normal persons in a disturbance of the relationship of the three factors, in the usually vagotonic reaction type and in the lability of all three factors. These differences are marked in catatonic stupors. The effect of drugs in the catatonic patient is similar to that in normal persons.

MALAMUD, Iowa City.

THE PATTERN OF SENSORY RECOVERY IN PERIPHERAL NERVE LESIONS. L. J. POLLOCK, Surg. Gynec. Obst. **49**:160 (Aug.) 1929.

In this communication are described the characteristic features of sensory loss in regenerating nerves. The material studied consisted of about 400 cases and included lesions of the ulnar, median, radial, brachial plexus, external popliteal and sciatic nerves. In a previous publication the author introduced evidence to show that the early shrinkage of the area of analgesia in the distribution of injured nerves was not due to the early regeneration of protopathic fibers but to the assumption of function by adjacent uninjured nerves. Contrasted with this observation he found in the present investigation that the pattern in actual regenerating nerves was strikingly different. Although the characteristic recession of the border of analgesia occurred, other changes were always present. A definite conception of these changes can be obtained by a study of the author's numerous charts. The same essential features were found in the various nerves studied.

The cases in which recovery was studied following neurolysis with resection and suture showed less return of pain perception than those in which the patient recovered spontaneously.

In conclusion, it is stated that the characteristic features of the sensory loss of regenerating nerves may be enumerated as follows: 1. Return of sensibility to pain, touch or temperature sense in that area of the sensory distribution of a nerve which is supplied by it alone, i. e., the isolated sensory area of the nerve. 2. Return of sensibility to pain, touch or temperature sense in patches some distance from the area supplied by an adjacent uninjured nerve. 3. Return of sensibility to pain, touch or temperature sense in deep indentations. 4. Diminution of degree of loss of sensation of pain, touch or temperature sense in the isolated sensory supply of a nerve. 5. Return of sensation of pain, touch or temperature in the border between the sensory supply of two nerves simultaneously injured. 6. Interlacing of the border of sensory loss of one type of sensation with that of another.

GARDNER, Cleveland.

THE RELATION BETWEEN CLINICAL NEUROLOGIC DIAGNOSES AND THE PATHOLOGIC ANATOMIC EXPLANATION OF DISEASE. KARL MAX WALTHARD, Schweiz. Arch. f. Neurol. u. Psychiat. **24**:231; **25**:89, 1929.

From a review of eighty-two cases personally studied at the Pathologic Institute of the University of Zürich, Walthard undertakes to determine to what extent a study of pathologic nerve tissue can be of assistance to clinical neurology. At the outset he warns that the anatomic changes represent an end-stage of a process that must be studied from a dynamic point of view. Even in conditions such as dementia paralytica, in which the clinical diagnosis and postmortem observations are generally in essential agreement, little is known about the true relation of clinical symptoms to tissue changes.

Numerous interesting cases are reported briefly. Spinal cords removed in a case of amyotonia congenita and one of pernicious anemia with unequivocal symp-

toms of involvement of the cord were entirely normal. In another case in which the patient was admitted in a demented state with a history suggestive of dementia paralytica but with negative serologic tests, a tumor of the cerebellopontile angle was found. Brains from several cases diagnosed as encephalitis showed nothing except hyperemia. The question as to what constitutes inflammation in the central nervous system is given careful consideration. The author discusses fully the proper technic of the study of nervous tissue and describes an ingenious card index system that gives the worker readily accessible information in regard to prepared and unprepared material available in the institute.

Walthard believes that present unsatisfactory relations between clinician and pathologist can be improved through the former stating the main questions at issue as well as furnishing a résumé of the history prior to the postmortem examination, thus enabling the latter to adjust his procedure to the needs of the particular case. He further urges that the clinician bear more clearly in mind the difference between reversible and irreversible changes, and that he study his cases from a psychobiologic point of view.

DANIELS, Rochester, Minn.

THE MIDBRAIN PITUITARY PROBLEM. W. W. MICHEJEW and E. M. PAWLJUTSCHENKO, Arch. f. Psychiat. **89**:271, 1930.

The authors discuss the relationship between the midbrain and pituitary gland with reference to the rôle played by them in diabetes insipidus. Five cases are reported; in three, diabetes insipidus was a prominent symptom, and in two, acromegaly. Case 1 was that of a man, aged 21, sexually underdeveloped, who had had diabetes insipidus for two and a half years before admission. This condition was more marked in the beginning than it was later. There was paralysis of the eye movement upward, with general rigidity and somnolence. The autopsy showed a tumor of the midbrain extending from the optic chiasm to the substantia perforata posterior. The pituitary body showed some atrophy. Case 2 was that of a man, aged 54, with diabetes insipidus. Here too, the condition was more pronounced at the onset. There was a gradual development of general weakness, involvement of the lung and tumor of the liver. An autopsy revealed cancer of the bronchi and metastases in the internal organs and pituitary gland. The latter had affected the infundibulum and had spread upward into the midbrain. Case 3 was that of a man, aged 21, with diabetes insipidus following encephalitis. The diabetes insipidus had gradually diminished under pituitary treatment. Case 4 was that of a woman, aged 52, with acromegaly for twenty-two years. There was some frequency of micturition but no increase in the quantity of urine. Case 5 was that of a man, aged 55, with a pituitary tumor and signs of acromegaly, but with no definite signs of diabetes insipidus.

In the five cases the author finds no proof of any direct relationship between pituitary disease and diabetes insipidus. He comes to the following conclusions: 1. Anatomic and experimental investigations show the presence of a close connection between the midbrain and the pituitary gland. 2. A complete destruction of the pituitary gland without disturbances of the midbrain does not cause diabetes insipidus. 3. In addition to diabetes insipidus, diseases of the midbrain may also call forth signs of dystrophia adiposogenitalis.

MALAMUD, Iowa City.

ANEMIC FOCI OF SOFTENING RESULTING FROM AN INFECTIOUS THROMBO-ARTERITIS OF THE LARGE BASILAR VESSELS. H. NATHAN, Ztschr. f. d. ges. Neurol. u. Psychiat. **123**:579, 1930.

Vascular changes in tuberculous meningitis have been observed for many years. Particularly significant are the focal changes seen in this form of tuberculosis. Often there is a well defined localization in the clinical picture, which cannot be demonstrated pathologically; just as often there are pathologic foci which seem to have given rise to no clinical signs. Among the foci of softening the hemorrhagic type is frequent, resulting from a tuberculous thrombophlebitis and thrombo-arteritis of the small and larger pial vessels.

In six cases of tuberculous meningitis, Nathan demonstrates that not only do the small and middle sized cerebral arteries show severe specific inflammatory changes, but that the large basilar arteries are concerned in the pathology. Up to now there has been no recorded case of anemic areas of softening due to the closure of a larger cerebral vessel. The inflammation of the vessel wall leads to the closure of the lumen by the formation of an inflammatory plug or by a marked internal proliferation as a result of the inflammatory stimulus. As a result of this closure of the vessel, there follows a disturbance in nourishment of the brain, and simple anemic softenings without evidence of inflammation. Clinical evidence of foci of softening are rarely found because they are covered by the more severe evidences of the meningitis. In one case, besides an alternately flaccid and spastic paralysis, there was a contralateral hyperhidrosis of the face. In this instance there was a thrombophlebitis tuberculosa of the superior longitudinal sinus. In five of the six cases, the patients were children under 1 year with evidence of miliary tuberculosis. The diseased arteries were the middle cerebral in three cases, the posterior cerebral in two cases, and both of these arteries in one case. Besides these there was a case of thrombo-arteritis of the internal carotid and middle cerebral arteries.

ALPERS, Philadelphia.

TUMORS IN THE CHIASMAL REGION. E. M. DEERY, J. Nerv. & Ment. Dis. **71**:383 (April) 1930.

A review is made of 170 cases of tumors in the interpeduncular region involving the optic chiasm in which transfrontal operations were performed. Of 225 transfrontal operations performed by Cushing at the Peter Bent Brigham Hospital, 55 were lesions not causing a chiasmal syndrome. Pituitary adenomas constituted 54 of 170 cases; seven of these cases showed hyperpituitarism and 43 showed hypopituitarism or transitional states. Pituitary dysfunction had long been present in these cases, but the chief complaint was due to the mechanical effect of the growth. Next in frequency are the craniopharyngeal pouch cysts, of which 47 are recorded. Two of the patients had attained the age of 62 years. In 41 of the cases the tumors pushed up from below, thus separating the optic nerves. Most of the patients in this category gave either laboratory or clinical evidence of pituitary insufficiency. Thirty-three showed definite sellar deformity. Next in order of frequency were the 16 cases of suprasellar meningiomas in which the primary complaint was failure of vision; ten cases showed normal roentgenograms of the sella. A special group of 15 patients showed negative results and the diagnosis was problematic; they all complained of headache and failing vision but operation revealed no abnormal conditions. There were 14 cases of glioma of the optic chiasm. The average age of the patients in this group was 14 years. Three of them showed general neurofibromatosis. Thirteen cases with a supposed chiasmal syndrome showed chronic localized arachnoiditis at the operation, all the patients having complained primarily of failing vision and headache. Four cases were found at operation to be third ventricle tumors. Aneurysm, cholesteatoma, sphenoidal ridge meningioma and angiomatous malformation are also reported by the author.

HART, Greenwich, Conn.

SEMIOLOGIC VALUE OF HEADACHES IN INFLAMMATORY INTRA-OCULAR DISEASES AND IN ORBITAL TUMORS. TEULIÈRES, Rev. d'oto-neuro-opht. **7**:615 (Oct.) 1929.

The majority of inflammations of the eye and its adnexa are accompanied by headaches—iritis, glaucoma and furuncle of the lids. This is true also of uncorrected errors of refraction. The two cases reported, in which headache was the prominent symptom, cannot be put in these categories.

Case 1: A man, aged 42, had suffered with constant violent headaches for fifteen months. After six months, a "flake" appeared in front of the right eye, and the patient had the sensation of a light veil over the left eye. The general physical condition was excellent. Both eyes showed light clouding of the vitreous;

visual acuity equaled 1 (feeble). There were no functional troubles of the musculature. The Wassermann reaction of the blood was negative; the nasal sinuses were normal, and antisyphilitic treatment was without effect. No indication of the cause of the chorioiditis was discovered.

Case 2: A woman, aged 66, fifteen days previous to consultation, had had sudden diplopia from paralysis of the left external rectus. Three days later, complete paralysis of the left common motor oculi appeared, accompanied by violent ocular, periorbital pains radiating to the left side of the head. No corneal anesthesia, changes in the eyeground or edema were observed. Vision in the left eye was 8/10. The right eye was normal. The Wassermann reaction was negative; roentgenograms of the sphenoidal fissure and of the nasal sinuses showed no opacities. In spite of this, antisyphilitic treatment was advised. No etiologic diagnosis was made in either case.

DENNIS, Colorado Springs, Colo.

THE RELATION OF THE ENDOCRINE GLANDS TO THE GROWTH AND DISTRIBUTION OF HAIR. Z. K. COOPER, Arch. Dermat. & Syph. **21**:1007 (June) 1930.

The distribution of the hair, as to both site and amount, is so easily studied that the establishment of any constant relationship between the pilary system and other functions of the body would be of great value. The endocrine glands offer the most promising field for this study. Cooper, reviewing the literature and conducting experiments of her own, establishes the following relationships: A reduction in parathyroid activity is associated with a scanty growth of hair; a reduction in pituitary function usually means loss of hair everywhere except on the scalp. On the other hand, hyperpituitarism is responsible for increase in the thickness and the area of distribution of the hair. No conclusive evidence has been presented to relate the activity of the thymus with the pilary system. It is known that hypotrichosis is associated with reduced function of the cortex of the suprarenal glands, and hypertrichosis with excessive function of this structure. With destruction of pineal activity early in life, abundant growth of the hair is the rule. There is much confusion about gonadal relationships in the modifications of the development of the hair, although it seems fairly clear that castration is associated with a reduction in the area of the hair and with a feminine distribution. There is an uncertain degree of hypertrichosis in diminished ovarian activity. In the case of the activity of the thyroid, the relative hairlessness of the cretin and the hairiness of the patient with acromegaly are well known. Cooper concludes with a regret that so little is known about normal variations in the growth and distribution of hair.

DAVIDSON, Newark, N. J.

A CLINICAL STUDY OF ONE HUNDRED AND THREE CASES OF SCLERODERMA. PAUL A. O'LEARY and RUBEN NOMLAND, Am. J. M. Sc. **180**:95 (July) 1930.

In 103 cases of scleroderma, in which the ages of the patients ranged from 3½ to 67 years, 48 were of the generalized type and 55 of the circumscribed type. Arthritic symptoms, mostly in the hands, occurred as the earliest manifestations in 58 per cent of generalized scleroderma. The next most common initial symptom was a vasomotor phenomenon in 33 per cent of the generalized group. The capillaries of the nailfold in the cases of generalized scleroderma without sclerodactylia appeared normal. Localized calcification was observed in 2 cases of this group.

General symptoms do not occur in circumscribed scleroderma. In the fifty-five cases there was morphea in thirty-eight, morphea guttata in three and scleroderma in the hands in fourteen. Occasionally morphea and facial hemiatrophy have been associated. Scleroderma is considered to be the result of a systemic disease rather than a localized disorder of the skin. The part played by the blood vessels in the production of the disease seemed an important one.

In differential diagnosis, chronic arthritis, Raynaud's disease, idiopathic cutaneous atrophy, dermatomyositis and Addison's disease must be considered. In most of the cases of both types of scleroderma, the basal metabolic rates were with normal limits. Chronic poisoning by arsenic could not be established as an etiologic factor. In generalized scleroderma the prognosis is poor, in the circumscribed type it is good.

MICHAELS, Detroit.

REPORT OF THE TREATMENT WITH MANGANESE CHLORIDE OF ONE HUNDRED AND EIGHTY-ONE CASES OF SCHIZOPHRENIA, THIRTY-THREE OF MANIC-DEPRESSION AND SIXTEEN OF OTHER DEFECTS OR PSYCHOSES AT ONTARIO HOSPITAL, BROCKVILLE, ONTARIO. W. M. ENGLISH, Am. J. Psychiat. **9**:569 (Nov.) 1929.

On the theory that certain modifications of salt metabolism would stimulate body defenses, English administered to several hundred patients a course of twenty or twenty-five intravenous injections of 0.25 per cent solution of manganese chloride. He was gratified by the results, which showed a decided physical improvement in half the cases and mental improvement in a third of them. In one case a catatonic patient who had had dementia precox for thirty years was mentally better. There was an average gain in weight of 12 pounds (5.4 Kg.) under this treatment. In discussing this paper, Dr. Norbury, of Jacksonville, Ill., pointed out the similarity between iron and manganese and considered that this work was part of a revival of interest in the time-tested pharmacology of a generation ago. Englander, of Newark, suggested that some of the improvement reported under manganese therapy might be due to the more intensive application of general nursing care, and that corresponding observations should have been made on patients similarly nursed but not treated with manganese. Englander also called attention to the possibility that chemotherapy, particularly in the field of colloidal chemistry, might have something to offer to psychiatry.

DAVIDSON, Newark, N. J.

THE PATHOLOGIC SIGNIFICANCE OF ACRO-ASPHYXIA. J. A. BEILIN, Med.-Biol. J. (Moscow) **5**:2:129, 1929.

Acro-asphyxia or cyanosis of the extremities and distal parts of the body, such as the nose, ears, etc., is found frequently either by itself or in connection with gastro-intestinal and cardiovascular systems. Often it is a clue to the pathologic processes within the organism and probably has some relation to the vascular pathology of the brain. Ten cases are described by the author with intensive studies of the various systems of the organism. The author's conclusions are as follows: Acro-asphyxia is accompanied by disturbances of sensibility of a nonradicular type. It depends on the disturbances of the vegetative nervous system, especially the sympathetic division and the endocrine factors. In certain stages of its development acro-asphyxia can lead to complete invalidism. The acro-asphyxia can be a result of various pathologic processes, not always clear, in various parts of the central nervous system. Temperature variations have nothing to do with the etiology of this condition. Following the diagnosis of acro-asphyxia, a thorough study of all factors which may have contributed to the condition must be undertaken; only then is rational therapy possible.

KASANIN, Boston.

STUDIES ON REGENERATION IN THE SPINAL CORD: IV. ROTATION ABOUT ITS LONGITUDINAL AXIS OF A PORTION OF THE CORD IN AMBLYSTOMA PUNCTATUM EMBRYOS. DAVENPORT HOOKER, J. Exper. Zool. **55**:23 (Jan. 13) 1930.

If a three myotome segment of the spinal cord of *Amblystoma punctatum* embryos is rotated 90, 135 or 180 degrees about its longitudinal axis, both the cephalic and caudal wounds will heal, provided the requirements for regeneration following simple section are fulfilled. If, at operation, the wound surfaces have

been properly apposed, the cells of the cord, the central canal and the fasciculi exhibit torsion to the extent necessary to provide continuity of each of these areas. This is true of 90 or 135 degree rotations, but does not hold for inversions. In inversions, or where the wound surfaces were not adequately apposed, fusion of the wound surfaces occurs, but the fasciculi of the cord take approximately the most direct course to corresponding fasciculi in the rotate, and vice versa. Such results indicate a high degree of regulatory capacity in the development of the fasciculi of the cord and emphasize the existence of a directional stimulus to neuraxial growth, whether bio-electric in nature or of another type.

WYMAN, Boston.

THE RÔLE OF PSYCHICAL FACTORS IN THE PRODUCTION OF ORGANIC NERVOUS DISEASE. A. M. RABINER and M. KESCHNER, J. Neurol. & Psychopath. **10**:311 (April) 1930.

The authors discuss the biphasic development of organic nervous diseases, first psychic and later organic, and suggest three possibilities as to the reason for this: (1) the functional phase may be independent; (2) the two phases may represent the same clinical entity, but the organic disease cannot be detected, owing to the grossness of technical methods, and (3) the functional condition causes organic changes in the brain. They give as evidence to show the influence of dysfunction on organic structures: (1) hyperdevelopment of nerve tissue under abnormal stimulation and (2) degeneration of central structures after injury of the nerve. They also point out relationships between certain functional conditions and organic diseases. They present three cases of psychic trauma that showed atypical, apparently functional, movements and later developed dystonia musculorum deformans — unfortunately a disease that is little known or understood.

ROBINSON, Kansas City, Mo.

OXYTHERMIA AND THE HIGH FREQUENCY CURRENT IN HEADACHES OF NASAL AND SINUS ORIGIN. SIEMS, Rev. d'oto-neuro-opht. **7**:599 (Oct.) 1929.

The author compares the efficacy of two modes of treatment for nasal and sinus headaches. The high frequency current was applied externally over the painful area and to the interior of the nose with a McIntyre tube. In oxythermia a stream of warm oxygen was directed to the inferior turbinate, the middle meatus and the spheno-ethmoidal recess by means of a cannula. Oxythermia acts on the nerve ends by three properties: warmth, and its bactericidal and stimulating action. The immediate effect is to produce an abundant flow of the intracellular serous infiltrate, to free the ostea and to evacuate the sinuses. A completely blocked nasal fossa is rendered permeable within twenty-four hours, the headache disappears and sleep is obtained. With the high frequency current, while the secretion is increased, the nose becomes blocked again toward evening, the headache is not relieved and the next day there is facial edema.

DENNIS, Colorado Springs, Colo.

TWO TYPES OF INTELLIGENCE. WILLIAM DÉRIAZ, Arch. de psychol. **22**:1 (Sept.) 1929.

Investigators have postulated two types of intelligence: the systematic and the empiric. The person with the first type approaches the solution of a problem by a survey of the total situation, the one with the second type immediately attacks the individual situation and proceeds in the solution by a trial and error method. The second type is inferior to the first. Dériaz has made a study of this concept by applying a series of tests to a large number of children. By the use of recording apparatus he tested quantitatively the ability to discriminate between weights, degrees of tactile stimuli and dimensions. To the same subjects he applied a number of intelligence tests and correlated these results with their school records. He concludes that the two types of intelligence exist and that each type is associated with distinctive motor and sensory reactions.

PEARSON, Philadelphia.

CUTANEOUS NEUROMA. JOHN B. LUDY, Arch. Dermat. & Syph. **21**:419 (March) 1930.

After reviewing the different meanings attached to the expression "neuroma cutis," Ludy suggests that it be limited to new growths containing abnormal nerve elements. The condition is rare, and when it occurs is usually diagnosed as a tuberculid. To avoid this frequent diagnostic error a biopsy should always be performed. In illustration, the author gives a case of his own. The patient was a middle aged colored woman who had an old tuberculosis of the chest. She had flat, pigmented lesions of various sizes, scattered over all four extremities. The masses were occasionally painful and always itched. Histologically, the areas consisted of a meshwork of nerve fibers in the corium supported by a fibrous stroma. No inflammatory infiltrate was found, although the blood vessels were collared by large round cells. Ludy believes that the nerve elements found in these growths represent proliferation from a parent trunk which subsequently disintegrates. He emphasizes traumatic and toxic factors as causative agents.

HEREDITARY NYSTAGMUS. WILLIS S. KNIGHTON, Arch. Ophth. **2**:437 (Oct.) 1929.

The type of nystagmus seen in multiple sclerosis, cerebellar disease and Bárány testing is "resilient"; that is, there is a quick component and a slow one. Hereditary nystagmus, on the other hand, is pendular; that is, the two components are equal. It represents an effort on the part of the patient to discover the best part of the retina for fixation. The line of descent may be either through both sexes, which is common, or through unaffected females, being in that case clinically limited to males. A case of the former type is presented, wherein the patient had hereditary nystagmus, as did his son and mother; the disease also afflicted five of the patient's nephews, but none of his brothers. Three male cousins also had this condition. This patient had had visual trouble since childhood; he also claimed to have hemophilia and night blindness, although clinical study showed no evidence of either. The refractive error in this patient was very small. He had pendular, horizontal nystagmus with a frequency of two tremors a second. Ophthalmoscopy suggested that he might also have retinitis pigmentosa sine pigmento.

EYE SYMPTOMS AND THE PARKINSONIAN SYNDROME. LEO J. GOLDBACH, Arch. Ophth. **2**:555 (Nov.) 1929.

In an effort to localize more accurately the optic signs of parkinsonism, Goldbach expresses an opinion that the upward and downward oculogyric crises are of metathalamic origin, that third nerve palsies are probably mesencephalic in site, that lesions of Toval's bundle cause lateral deviation and that the little understood body of Luys may also, in disease, be responsible for lateral deviation. He warns against the danger of assuming the existence of a brain tumor in cases of optic neuritis or of diagnosing cerebrospinal syphilis in the presence of an Argyll Robinson pupil, pointing out that both of these may be seen in chronic encephalitis.

HOMONYMOUS HEMIANOPSIA DURING PREGNANCY AIDED BY REFLECTING PRISM. CHARLES A. YOUNG, Arch. Ophth. **2**:560 (Nov.) 1929.

Presenting a case of homonymous hemianopia in a woman who was pregnant, this symptom having come on suddenly during the eighth month of gestation, Young speculates as to the cause. He considers three possibilities: hysteria, the pregnancy and a circulatory disorder. Rejecting the first two, he concludes that vascular spasm was responsible. By using a prism that reflects objects on the blind side obliquely into the functioning side of the retina, Young achieved sufficient visual return so that his patient could drive an automobile.

DAVIDSON, Newark, N. J.

HUNTINGTON'S CHOREA WITHOUT CHOREIFORM MOVEMENTS. DESMOND CURRAN, J. Neurol. & Psychopath. **10**:305 (April) 1930.

Curran reviews the semiology of Huntington's chorea without choreiform movements and reports carefully a patient's condition and the family history. He points out a direct connection between the condition of the patient and that of other members of the family. Five other members of the family had this disease, including the patient's mother, and it is developing in one son of the patient. Except for dysarthria, there were no motor disturbances. The complaints were all objective mental disturbances. Curran concludes from the mental disturbances and the family history that the condition is Huntington's chorea.

ROBINSON, Kansas City, Mo.

DECREASE IN INTELLIGENCE WITH INCREASE IN AGE AMONG INMATES OF PENAL INSTITUTIONS. GEORGE REX MURSELL, J. Juvenile Research, **13**:197, 1929.

Mursell collected the results of the Kuhlman-Binet individual mental examinations and the Kuhlman-Anderson group examinations given to inmates of Minnesota penal institutions. He found that the average intelligence quotient decreased after the age of 40. There may be several reasons for this: (1) senile or arteriosclerotic dementia may occur; (2) delinquents who have low mentality in youth may continue to be delinquent in old age; (3) the lack of educational opportunities when the group now over 40 years old was young. He recognizes that he examined a selected group so that the results cannot be considered representative of the general population.

PEARSON, Philadelphia.

THE IMPORTANCE OF A CLINICO-ETIOLOGIC CLASSIFICATION OF HEADACHES. J.-A. BARRÉ, Rev. d'oto-neuro-opht. **7**:607 (Oct.) 1929.

The number of causes of headaches is so great that the difficulties of tracing them to their source are extreme unless various methods of study are adopted. All headaches have been, for the most part, regarded as being alike in character. On the contrary, there are differences that give each kind a distinct physiognomy. The author believes that "the cause is engraved in the symptom, and that each particular cause gives rise to a symptom with a little special form." Headache should be studied in minute detail.

DENNIS, Colorado Springs, Colo.

THE VALUE OF BLOOD BILIRUBIN ESTIMATIONS IN THE DIFFERENTIAL DIAGNOSIS OF CEREBROVASCULAR ACCIDENTS. GARNETT CHENEY, Am. J. M. Sc. **180**:24 (July) 1930.

Because of the difficulty in diagnosing the different causes of cerebrovascular accidents, the application of the blood bilirubin tests seems interesting. The author concludes from a series of fifty cases that a definite increase in the icteric index with a negative direct van den Bergh reaction following a cerebrovascular accident usually indicates that a hemorrhage has occurred, and that a normal index after the first day is strong evidence against a hemorrhage.

MICHAELS, Detroit.

THE EFFECT OF TEMPERATURE UPON THE ABSOLUTE REFRACTORY PERIOD IN THE NERVE. WILLIAM R. AMBERSON, J. Physiol. **69**:60 (March) 1930.

In order to observe the effect of temperature on the duration of the absolute refractory period in the nerve, Amberson carried out a group of determinations in the sciatic nerves of large *Rana esculenta*. Over the range examined (from 0 to 27 C.) the experimental values obtained outline a curve which is approximately exponential in form. The Q_{10} is 3.06. A review of the literature covering the work done in this field is given.

ALPERS, Philadelphia.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, May 23, 1930

WILLIAMS B. CADWALADER, M.D., *President, in the Chair*

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS WITH SOME UNUSUAL FEATURES POSSIBLY DUE TO "EXHAUST GAS INTOXICATION." DR. JOSEPH C. YASKIN.

This case shows: (1) a primary involvement of the lower bulbar nuclei, (2) a rapid and diffuse implication of almost the entire musculature and (3) some possible occupational, etiologic factors.

Clinical History.—J. D., aged 42, was admitted to the Graduate Hospital (service of Dr. T. H. Weisenburg) on April 25, 1930, complaining of difficulty in talking. The family and past medical histories were irrelevant. The patient had been married for twenty years and had nine children in good health. He was engaged in the garage business, and during the past winter to a great extent was confined within the garage with the doors closed, thus being exposed to a large amount of "exhaust gas." Four months prior to admission, he observed that he was unable to talk properly, his tongue becoming "thick." He felt as if he had a continuous cold in the nose. He could start and stop talking whenever he pleased, but had difficulty in pronouncing certain words and numbers, such as "three." Apart from this complaint, he had had no difficulties; there was no history of any infection or of diplopia, headache, vertigo, tinnitus or symptoms referable to the cardiovascular, respiratory, gastro-intestinal or genito-urinary systems. He complained of no weakness in the arms or legs nor of any sphincter difficulties. There was no history of any serious loss of weight, and sleep was undisturbed.

Physical Examination.—The patient was fairly well developed and nourished; the skin was somewhat sallow; there were a few carious teeth, and the tonsils were enlarged. The thyroid was not enlarged; the heart and lungs were normal; the blood pressure was 140 systolic and 80 diastolic; the pulse rate was 80. The abdomen presented no masses or tenderness.

Neurologic Examination.—The sense of smell was intact. The ocular fundi and fields were normal. The pupils were equal and regular and responded normally to light, distance, convergence and concentric stimulation. There was no ptosis of the lids, and all extra-ocular movements were normal. There was no nystagmus. The muscles of mastication were normal, and there were no sensory disturbances over the face. There was definite weakness of the facial muscles when the patient attempted to whistle, and weakness of the buccinator and orbicularis oris was shown in puffing out the cheeks. There were tremors of the lower part of the face, but neither weakness nor tremors of the upper part. The sense of taste was normal on both the anterior and the posterior parts of the tongue. There was no disturbance in hearing, and Bárány tests (Dr. Lewis Fisher) revealed no abnormalities in the vestibular mechanism. There was slight weakness of the soft palate, and fibrillary tremors were observed on the right side of the palate. Examination of the larynx (Dr. Gabriel Tucker) showed: "Gross motility of the larynx is normal. There is possibly a slight impairment in abduction, and the epiglottis is possibly a little narrower than normal. There is marked relaxation of the cords. There is a tendency for the cords to approximate first at a point just at the anterior end of the vocal processes, the cords being crescentic immediately anterior to this. The patient is still able to change the pitch of his voice but with considerable effort. His larynx seems to close sufficiently to prevent

aspiration of secretion." The patient had no difficulty in swallowing. The speech was somewhat nasal. He had difficulty in pronouncing certain words, such as those requiring the combined use of the tongue and lips. The functions of the sternocleidomastoid and trapezius muscles were intact. The tongue was decidedly diminished in size, protruded only slightly, was greatly wrinkled, showed marked fibrillary twitchings and was soft and spongy on palpation. There was practically no weakness or disturbance in tone in any of the groups of muscles of the trunk or extremities, and yet there were pronounced fibrillary tremors involving both shoulder girdles, the arms and the muscles of the back and of the gluteal region, while the electric reactions were normal. The abdominal and cremasteric reflexes were intact; the biceps, triceps, knee and achilles reflexes were symmetrically exaggerated, and there were no pathologic reflexes. Deep and superficial sensibilities were intact throughout, and there were no disturbances in synergic control. Roentgenograms of the skull and spinal column showed no abnormalities.

Laboratory Data.—Repeated examinations of the urine gave entirely normal results. A blood count revealed: erythrocytes, 4,250,000; hemoglobin, 85 per cent; leukocytes, 10,000; the differential count gave: polymorphonuclears, 68; small lymphocytes, 25; mononuclears, 6 and eosinophils, 1 per cent. Chemical analysis of the blood showed: sugar, 82; urea nitrogen, 15; chlorides, 0.59 mg., and calcium, 9.8 mg. per hundred cubic centimeters. The Wassermann test of the blood gave negative results. The test of the spinal fluid revealed: pressure, 8 mm. of mercury with a normal rise on compression; 7 cells per cubic millimeter; protein and sugar content normal; colloidal gold curve flat; Wassermann reaction negative.

Course.—The patient showed very few changes with rest, elimination by hot packs, etc., and was discharged from the hospital on May 10. When reexamined a month later, there was neither improvement nor progression of the symptoms.

Comment.—The anatomic diagnosis is based on a serious involvement of the nuclei of the hypoglossal and the facial nerves and to a lesser extent of the nucleus ambiguus, of the spinal anterior horn cells and of the pyramidal tracts. The paralysis, tremors and atrophy of the tongue are almost equalled by those of the orbicularis oris and of the buccinators. The nuclei of these are in close anatomic proximity to those of the hypoglossal nerve, whereas the other components of the facial nerve (Purves-Stewart, Jones: The Diagnosis of Nervous Diseases, ed. 6, New York, E. B. Treat & Company, 1924, p. 220), which are remotely located, have so far escaped. While chronic progressive spinal muscular atrophy cannot be definitely ruled out, by far the more likely diagnosis is amyotrophic lateral sclerosis. Another interesting feature is the onset with lower bulbar symptoms and particularly the symptomless, rapid, diffuse implication of a great many muscle groups. This, in itself, is suggestive of an underlying toxic condition. The possibility of the "exhaust gas," to which the patient had been constantly exposed during many winter months, as an etiologic factor naturally presents itself. A search through the literature gives no information on this subject.

A New Staining Method for Fresh Brain Tissue. Dr. S. DeW. Ludlum and Dr. A. E. Taft.

In studying fresh brain tissues, we have been working with a new method of staining. The technic has already been described (*Proc. Soc. Exper. Biol. & Med.* **27**:582, 1930); it consists in putting a small piece of brain tissue into a 10 per cent solution of mild silver protein for from twelve to twenty-four hours. Small fragments are then removed and washed only enough to remove the excess stain, and are placed on an ordinary glass object slide with a thin coverslip, which is pressed lightly in order to form a thin film. This method offers an opportunity to examine tissue that has not been subjected to fixatives and dehydrating fluids. In the microscopic field one sees the unstained granular intercellular substance on which cells and vascular elements appear in brown or black as a background. As shown in the photographs, the cell outlines are distinct, including the processes of the different types of cells.

For anatomic purposes, the method shows the entire cell picture, without the disfiguring result of sectioning. It also gives unusual pictures of cell groups and extensive vascular ramifications. For pathologic study, it offers a complete opportunity to see such changes as lipoid degeneration, cell shrinkage, arterial changes, softening and cell proliferation.

The photographs show, in addition to normal appearances, the presence of satellite cells and an increase in oligodendroglia. Pictures from young rat's brains also demonstrate young forms of nerve cells. The granular intercellular substance is especially well portrayed.

This method is useful as a supplementary means of study, in connection with fixed and embedded material which preserves the relations of the tissue elements. Aside from this feature, the silver stain of fresh tissue affords a complete method of study, and has the advantage of allowing prompt examination.

Quantitative Pupillary Light Reflex. Dr. Louis Lehrfeld.

This article appears in this issue of the Archives, p. 159.

Involvement of the Nervous System Following Extraction of Teeth. Dr. Wilbur Bailey.

Clinical History.—A woman, aged 66, seen in the neurologic outpatient department on Dr. Spiller's service, had been perfectly well until Feb. 16, 1929. At that time she had six teeth extracted. One week later, she had six more teeth extracted and finally, several days later, she had had the remaining seven removed. On that day, when she came home she had difficulty in speaking and moving the jaws, and after about two more days she became speechless and difficulty in swallowing developed. This condition persisted until April, 1930, without any marked change in the symptoms.

Examination.—The patient was able to understand conversation perfectly and seemed of normal intelligence. She complained of severe pain on both sides of the face, especially on the right. She also complained of severe headache.

At that time Dr. Spiller made the following report: "The patient is unable to pucker her lips, and blows out a lighted match with great difficulty. She shows no weakness on closure of the eyelids on each side. The tongue can be protruded slightly, but not beyond the lips. It shows marked atrophy along the edges and fibrillary tremors are distinct. The soft palate is innervated very imperfectly. The patient is entirely speechless and at most can make only a few unintelligible noises. Her understanding of speech is apparently normal. When a glass of water is given to her she takes a little into her mouth and swallows it with great difficulty while a great deal of the water escapes and leaks out over her lower lip. The contraction of the masseter muscles is feeble."

Comment.—Whereas this case seems to be the only one in which a bulbar palsy has followed the extraction of teeth, facial paralysis following the extraction of a tooth is no novelty. In 1878, J. S. Nairne (*Brit. M. J.* **1**:335, 1878) described the first case of facial palsy following extraction of a tooth, and in 1896, H. von Frankl-Hockwart (*Wien. klin. Wchnschr.* **10**:145, 1896) collected six such cases from his own clinic. Up to the present, twenty-four cases have been reported in the literature in which a facial paralysis has followed extraction of teeth. In addition, seven cases have been found in which a facial paralysis occurred following or coincidental with dental caries, difficult eruption and swelling around the teeth (Kron, Hermann: Sammlung von Vortragen aus dem Gebiete der Zahnheilkunde, Leipzig, 1912, no. 12).

Von Frankl-Hochwart suggested that the micro-organisms found in the pus at the apexes of extracted teeth were widely disseminated during the extraction and were given an excellent field for propagation. He therefore proposed that the facial palsy was probably the result of infectious or toxic factors.

Orr and Rows (*Rev. Neurol. & Psychiat.* **8**:721, 1910) wrote a paper on "The Histological Evidence that Toxins Reach the Spinal Cord Via the Spinal Roots."

They placed a sealed celloidin capsule containing a broth culture of an organism under the gluteus muscle of dogs or rabbits in direct contact with the sciatic nerve. After the capsule had been in apposition to the nerve for ten or twenty days, the animal was killed, and sections of the nerve were made. The sections obtained in this manner showed a marked inflammatory reaction in the proximal part of the nerve, which became progressively milder as the distance from the source of toxin increased. The products of inflammation were found to be chiefly perivascular in location and in the lymphatic spaces of the nerve. Orr and Rows concluded definitely, therefore, that they were correct in their belief that the lymph stream in nerves is an ascending one. They further expressed the opinion that toxins could be carried to the cord by this path.

It is not difficult to believe, in view of the large area that was exposed in the case that has been described, that perhaps some of the toxins that were carried centrally along the lymphatics were responsible for the ensuing bulbar paralysis.

EPILEPSY VERSUS DIABETES. MAX TRUMPER, PH.D.

The author thinks that in epilepsy and diabetes the fundamental biochemical mechanisms are, in part at least, antagonistic. Diabetes is associated with ketosis and anhydremia; epilepsy, on the contrary, is characterized by alkalosis and hydremia, precipitating a seizure, and is benefited by therapy designed to produce a state of ketosis and dehydration. From the standpoint of the biochemical mechanisms involved, the incidence of epilepsy in diabetes should be extremely rare since ketosis and dehydration, which are prominent features of diabetes, constitute the therapeutic procedure in the prevention of seizures. Clinical experience appears to confirm this theoretical observation. Joslin said that he was able to find no clearcut case of epilepsy among 5,086 true cases of diabetes. He mentioned that Geyelin had knowledge of two diabetic patients with epilepsy, but did not state which condition was primarily present nor the severity of either condition. If an epileptic person is known to have diabetes, one must be guarded in using the ketogenic diet and restricting intake of water for the control of convulsions, since the deliberate production of a ketosis superimposed on the already existing state of ketosis that accompanies diabetes may have serious consequences. A study of such a person will give a clearer insight into the factors that are operative in the causation and control of epilepsy.

DISCUSSION

DR. MAX TRUMPER: I am convinced of the fundamental contribution of Dr. Fay's research on the value of marked water deprivation therapy in convulsions of epilepsy. One reason why we have been so confused in making a differential diagnosis, as well as in understanding the biochemical mechanism of convulsions, is that the observations of blood chemistry have been misleading and have complicated unnecessarily the clinical picture. For example, the literature is full of contradictory reports on the question of blood sugar in the convulsions of epilepsy, eclampsia, etc. One reason for this has been a neglect of the state of carbohydrate nutrition at the time the blood specimens were obtained. Everyone knows the necessity of obtaining specimens of blood for chemical analysis at the fasting level, but this is frequently neglected in studying blood sugar levels during or after a convulsion. Observations are reported without mentioning the method used and more especially failing to qualify the figures with data as to the state of nutrition of the patient.

THE SYNDROME OF AMYOTROPHIC LATERAL SCLEROSIS IN EPIDEMIC ENCEPHALITIS. DR. A. M. ORNSTEEN.

Clinical History.—O. H. F., a man, aged 45, married, in November, 1928, contracted a cold which lasted about a week. His entire body ached and he had mild symptoms in the upper respiratory tract and occasional chills, but continued

to go to work each day, although he was extremely weak. After recovering from the cold, his wife noticed that at the end of his day's work he appeared to be somnolent and yawned frequently. The drowsiness continued into December, and during this time he had amblyopia and diplopia on many occasions. The somnolence had not yet worn off entirely, when, in the latter part of December, the speech became thick. He thought that the tongue was swollen. The speech became thicker during the following two months, and he began to have some difficulty in swallowing. Liquids were regurgitated into the nasopharynx, and he frequently "choked" when swallowing. Dysarthria and dysphagia became progressively and rapidly worse. In the spring, 1929, twitching of the muscles in various parts of the body was first noticed and in June he began to have weakness in the right lower limb sufficient to cause the forepart of the foot to flop in walking. Since then all of the symptoms had become aggravated. Speech became less distinguishable, saliva drooled freely and swallowing became so difficult that much effort and care were required to complete a meal, the diet becoming more selective on account of this. The gait had become spastic, especially in the right lower limb; the muscular twitching was more active and constant in all parts of the limbs and trunk.

Physical Examination.—Both lower limbs were affected, the right much more than the left, the former being used as in a hemiplegic gait. There was no ataxia in the gait, no Romberg swaying. Both lower limbs were weak; the knee and ankle reflexes more bilaterally hyperactive on the left than on the right side; there was a definite Babinski plantar response on the left and an extension of the small toes with a motionless great toe on the right. Unsustained clonus of the patella on the right and of the ankle on the left were present. The abdominal reflexes were active on both sides. The tendon reflexes of the arms, as well as the radial periosteal reflex, were hyperactive; bilateral Hoffmann reflexes were present. The patient could use the upper limbs normally but with poor power.

The entire skeletal musculature was wasted, particularly the proximal segments of the upper limbs and shoulder girdle. The small muscles of the hands were moderately wasted. The thighs and calves were more wasted on the right than on the left side, and the glutei were not normal. The facial muscles were distinctly atrophic as evidenced by thin lips, sunken cheeks and flattened masseter and temporal masses. Motor weakness was readily recognized in all the wasted muscles. The patient could not pucker the lips or puff out the cheeks, nor could he move the lower jaw laterally or fully depress the mandible. The biting power of the masticatory muscles was defective. The tongue was completely paralyzed, atrophic and fibrillating; he could not move it about in the mouth or protrude it. In the recumbent position the tongue fell back and embarrassed the patient's breathing. The soft palate was motionless in attempted innervation and there was no gag response; a feeble gag reflex was produced by touching the pharynx. Laryngeal examination revealed complete paralysis of the right arytenoid cartilage and vocal cord. Movements of the frontalis and orbicularis oculi muscles were better than those of the orbicularis oris and buccinators, which is usually observed in bulbar palsy. Fluoroscopic examination showed a freely movable diaphragm with full excursion on each side. The electric reactions of the wasted muscles were generally those of quantitative reduction in excitability to both currents. In no muscle was there a complete degenerative formula. In the more affected groups, the faradic response was wanting, while the galvanic reaction was diminished with no modal or polar changes.

A striking aspect of the picture was the generalized myoclonia and fibrillation. In the masseters, lips and hands, the twitchings were small and of the nature of fibrillation. In the neck, arms, legs, thorax, buttocks and abdomen, the movements were intense and involved large portions of muscles so that they appeared rather to be myoclonic contractions than fibrillary twitchings. The motor unrest was continuous and was observed at various times in every muscle from the masseters down to the achilles tendons. The small muscles of the hands fibrillated, but fibrillation was not observed in the small muscles of the feet.

Sensation was carefully tested in all dermatomeres and in the areas of the cranial nerves without finding a deficiency in any form of sensory perception. The pupils, extra-ocular muscles, eyegrounds and visual fields were normal. Serologic tests gave negative results in the blood and the spinal fluid; the latter contained 2 units of protein (normal, 1.5 units) and 8 lymphocytes; the chloride and sugar contents were within normal limits. Manometeric observations were normal. A general visceral survey revealed nothing of note.

PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, May 16, 1930

FREDERIC H. LEAVITT, M.D., *President, in the Chair*

PSYCHOANALYTIC METHODS IN RELATION TO CHILD AND ADOLESCENT GUIDANCE. DR. B. SACHS (by invitation).

I desire first to express my opinion that the further progress of psychiatry, especially in America, does not lie solely along freudian lines, and that the national mental hygiene movement will not be promoted by overemphasis on psychoanalytic doctrines. The psychologist may have reason to accept these doctrines in toto, but psychiatrists should adopt them only after careful logical scrutiny. I believe that psychoanalysts may be charged chiefly with a lack of logical faculties; all of them are guilty of generalizations from insecure and unproved premises. While they have revealed many interesting features regarding dreams, the unconscious, infantile fixations, and the like, there is no reason to infer that everything that had been established before the freudian era is to be discarded. Freud himself acknowledged that there might have been mistakes in some of his doctrines, but his followers seem to take everything for gospel truth.

Several books have recently appeared on the subject of psychoanalysis, which give everything in favor of psychoanalysis in tremendous detail and fail to cite a single argument, among the many that have been advanced, against psychoanalytic doctrines. I refer to the latest contribution as the "Bible," with the full text on one page and annotations on the opposite page. As an example of the criticism which should be applied to all freudian doctrines, I would refer to the Oedipus complex; there is reason to doubt whether the Oedipus complex plays anything like the rôle ascribed to it by many freudians. Freud, himself, expressed doubt as to whether the Oedipus complex applies at all to normal persons; but every one of his followers, in this country at least, seems to speak of it as though it were established beyond shadow of a doubt.

In illustration, I cite the case of a boy, aged 14, in which there is need of solving the problem in a psychologic rather than in a psychoanalytic way. The Oedipus complex surely played no part in this case, in which there was a distorted relation between the boy and his parents. The chief evil done by psychoanalytic doctrines is that matters of much greater importance are often overlooked and that freudian followers who are obsessed by their own doctrines seem to miss the more important factors that a case presents. This is especially true in solving the problems presented by young girls. I charge psychoanalysts especially with an utter disregard of many factors that go to the development of character, at the expense of finding some latent sex factor which may be of academic interest, but has little, if anything, to do with the future development of character.

I plead that undue importance should not be attached to freudian doctrines, especially in the development of child guidance clinics, for the simple reason that other and much more important factors may be overlooked. I refer particularly to the excellent "Knighthood of Youth" movement in the public schools of New York, which fortunately has not yet received the slightest freudian taint, and which has been doing much more in development of character and good citizenship than could possibly be hoped for by the application of freudian doctrines.

Psychologists and neurologists have a serious duty in keeping the community sane on many such questions, and it is not wise to let freudian disciples cry out from the housetops while we merely sit by and let them do the talking. This gives the public the impression that an oracle is speaking and that there is no gainsaying the great truths that are being uttered. I am of the distinct opinion that much of the harm done by psychoanalysis would be neutralized if psychoanalytic investigation and psychoanalytic treatment of youthful persons were conducted by physicians of the same sex. Girls should be analyzed by women, and boys by men. Even so, men and women who analyze must be of the strongest moral fiber.

DISCUSSION

DR. SAMUEL LEOPOLD: No one doubts that the psychoanalytic movement has given one approach to psychiatry that cannot be forgotten, and that is the dynamic approach. As a matter of research, I believe that the genius of Freud has given to psychiatry much that is valuable. I am not in accord with some of the theories of psychoanalysis; I am not in sympathy with the Oedipus theory of an adolescent; neither am I in sympathy with an attack on normal persons by an approach purely to the unconscious. I believe that one should never lose sight of the fact that there are many ways of approaching and successfully attacking individual behavior problems. Nevertheless, one must not forget the importance of the dynamic approach that psychoanalysis has provided. The facts that a child needs to break away from the family situation and that failure to do this may create a serious breakdown have been revealed by psychoanalysts. I agree with Dr. Sachs that it is not necessary, and I believe that it is rather dangerous, to approach the adolescent with the psychoanalytic procedure alone. I believe that there are not more than 2 per cent of adults who would benefit more by psychoanalysis than by any other procedure.

On the question of sex, however, I differ with Dr. Sachs. I believe that the child begins to show sex manifestations very early in life, and it is a mistake to shut one's eyes to this important fact; it is essential to develop a more natural approach than hitherto has been given.

DR. CHARLES K. MILLS: Dr. Sachs and I are old colleagues and comrades in the American Neurological Society and other medical associations. He and I were among the first to take the view that I have always believed to be the correct one with regard to the question of psychoanalysis and the methods of investigation used by psychoanalysts. I have studied the views of Freud and his close followers, and I believe that they have done much harm, although possibly a little good, also. I think that my views could not have been better expressed than by Dr. Sachs.

DR. JOSEPH C. YASKIN: Dr. Sachs' paper was interesting and it would be difficult for him to find a more agreeing audience than is to be found in Philadelphia. So far as the psychoanalytic movement is concerned, there is no ground more barren than Philadelphia. Not only is there no definitely organized effort to develop this new addition in the healing art, but there is indeed a very active opposition on the part of the older men who are the natural leaders in neuropsychiatry in this community. The impetus given by S. Weir Mitchell in the treatment of the neuroses and psychoneuroses and the methods employed by him are still followed to a large extent. It must be admitted that although some of the psychoneuroses will yield to physiologic rest and hyperalimentation, while others will improve, temporarily at least, under suggestion, persuasion, reeducation, etc., yet a great many cases remain uninfluenced by these methods of approach. The reasons for this are not so difficult to understand when one remembers that almost all the older methods, as applied to the majority of cases, are only means of temporizing. Certainly orthodox neuropsychiatry explains neither the etiology nor the structure of the neuroses, and consequently cannot offer an honestly natural, scientific therapy. There is a strong feeling in some of us, as there has been in Dr. Sachs for many years, that this branch of neuropsychiatry requires modification. I believe that psychoanalysis offers the best method at the present time.

It has been stated this evening that psychoanalysis is most unscientific, that it is based on vague assumptions, fancies and mysticisms, and that its methods are expensive, time-consuming and impractical.

In reply, I say from experience with both methods, that psychoanalysis is no more unscientific than the rest of neuropsychiatry or medicine in general. There is one factor that critics of psychoanalysis usually forget — that one, himself, must be analyzed in order to have the proper understanding of psychoanalysis. One not familiar with the technical difficulties of biochemistry and electricity is certainly in no position to offer criticisms in these highly specialized branches. Those of us who have been analyzed believe that psychoanalysis gives the best approach to an understanding of the mechanism of the neurosis, and what is more important, to the why and the how of the neurotic constitution. Psychoanalysis points the way of the evolution of the psyche in its relation to the development of the race, family and individual, as well as of the neurosis. Its application to the child problem, in time, cannot be without considerable value from both a prophylactic and a therapeutic standpoint. It takes into account the unconscious motivation that, in a large measure, dominates our ways of feeling, thinking and acting, which cannot be explained by older methods of investigation and treatment. It is conceded that the method is cumbersome, but this is true of every new scientific procedure that becomes easier and simpler in operation in time. Lastly, psychoanalysis at the present time at least, is not intended to be a routine method for the treatment of psychoneuroses and behavior disorders. There are clear indications and contraindications in the selection of cases for analysis — conditions that are applicable in other rational modes of therapy.

Dr. Everett S. Barr: I have been impressed with Dr. Sachs' paper; I believe it is thoroughly in accord with the feeling in Philadelphia. I think that one of the most important things mentioned by Dr. Sachs is perhaps, the substitution of a psychologic study for so-called psychoanalysis. This is explained by the fact that psychoanalysis gained much popularity by providing a rational approach to many problems of psychoses and neuroses which were entirely unexplained before the advent of the freudian philosophy. Whether this explanation is true or not, it has been greedily seized on. I believe that many psychiatrists and many laymen are aware of the dangers of psychoanalysis, and that there is already a growing opposition to it. Dr. Sachs spoke of the cleavage between psychologists and psychiatrists, saying that psychologists had seized on psychoanalysis to entrench themselves more strongly. As I have often stated, I think it is important that psychologists and psychiatrists should work together, not allowing their work to overlap. The pathologic should belong to the medical profession, that is to psychiatry.

Dr. Max Levin: Dr. Sachs confined himself almost entirely to a discussion of the rôle of the Oedipus complex in psychoanalysis. It is unfortunate that this was made necessary by time limitations; it must be remembered, while the Oedipus complex is one of the principal elements of psychoanalytic theory, that it is by no means the only element. Equally if not more important is the contribution made by psychoanalysis in the direction of demonstrating the unconscious motivation of behavior, particularly of neurotic symptoms.

Dr. Sachs spoke of his common sense approach in his cases, by which he apparently means an appeal to the conscious sentiments of the patients — an appeal to their desire for approval, to their reasoning powers, etc. Only a cursory experience with neurotic and maladjusted persons ought to convince one in most cases that only a small portion of the symptoms can be understood on a common sense level. Does one not often meet patients who cannot understand their symptoms, who are annoyed by their absurdity and yet cannot shake them off? I recall an intelligent patient who complained that when he read, he felt impelled to stop at the end of every line and count the commas; he could not understand why such an apparently foolish symptom should arise in him; yet it annoyed him exceedingly. Freud and his followers have shown that many of these neurotic symptoms occur as a result of causes of which the patient is not conscious; and

it is to their credit that they have greatly clarified in this way many puzzling problems in psychopathology.

Since Dr. Sachs implies that psychoanalytic theory has little to do with common sense, it may be worth while to scrutinize a little more closely the methods involved in the common sense approach. Among these methods one finds an amazing array of therapeutic procedures such as suggestion by means of electric shocks, nauseous medicinal mixtures given with the assurance that "this is going to cure you" and the like. I fail to see how one can assume that these methods are based on a greater measure of common sense than the methods of psychoanalysis.

DR. HENRY I. KLOPP: At the Allentown State Hospital, we try to instil in the minds of the young people the importance of character building that, in principle, is carried out in their daily routine activities. In evidence of this, the president judge of one juvenile court has favorably commented on our work in propounding the question: "Why is it that when we send children to the State Hospital they do not become repeaters, whereas, those committed to reform schools too frequently become recidivists?" Naturally, these cases are all studied from a psychiatric point of view and instead of emphasis being placed on punishment and discipline, treatment and character formation are stressed. We do not make use of psychoanalysis with these children. In a great majority of those who come to the hospital, particularly for observation, diagnosis and treatment, the history shows clearly the underlying factors, most important of which is environment, although heredity cannot be entirely ignored. In a small percentage of adult cases psychoanalysis is of benefit.

DR. B. SACHS: I acknowledge the many interesting and valuable facts revealed by Freud and some of his followers, but I must insist that such observations should not be accepted as gospel truth; they should be met with the same logical scrutiny that is applied to other scientific doctrines; above all, psychiatrists should be careful not to draw broad generalizations from insecure premises.

DEVELOPMENT OF A CHILDREN'S INSTITUTE FOR MENTAL HEALTH. DR. HENRY I. KLOPP.

Dr. Klopp's presentation (*Mental Health Bulletin of the Department of Welfare,* State of Pennsylvania, vol. 8, no. 1) covered the new development of a children's unit in the Allentown State Hospital. He described the new work in detail, pointing out the needs for the Children's Institute for Mental Health, and what work led to its formation. A description of the new unit and its function included details of the building and lay-out, purposes and function, type of children, statistics, care and treatment, reeducation program, through the school, social service work and rehabilitation, research and development of the plan during its function, with a final summarization of the relation of the Children's Institute to the community.

DISCUSSION

DR. B. SACHS: I am much interested in what Dr. Klopp is doing. It seems to me that it is excellent work, particularly since he is also giving attention to the development of character. I was interested to see that he had a great many postencephalitic psychoses. I am glad to know of this work, and I think that he is to be congratulated on the report he is able to make.

DR. B. SACHS: Is this a public institution?

DR. HENRY I. KLOPP: Yes, a state hospital for mental diseases.

DR. B. SACHS: Do you make charges?

DR. HENRY I. KLOPP: A patient can be admitted to the hospital on a so-called private rate basis at not more than $10 per week. The majority of patients are in poor financial circumstances; they come on endorsement of the Poor Directors of the county in which the parents have a legal settlement in Pennsylvania. For those committed by court order, the actual cost, approximately $7 a week, is authorized. Frequently the relatives are able to pay this.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

May 15, 1930

E. W. TAYLOR, M.D., *President, in the Chair*

CENTRIPETAL PATHWAYS OF VISCERAL AND VASCULAR PAIN. DR. E. A. SPIEGEL, Vienna.

It is possible to produce pain by dilation of the wall of the aorta (experiments in collaboration with Dr. Wassermann). Such a dilation can be produced by the injection of sodium chloride solution in a sac of aortic wall isolated from the arch or by introducing a special apparatus (dilating springs) in the ascending part or arch of the aorta. A drug which contracts smooth muscle, such as barium chloride, and thus produces pain in other arteries, cannot do so in the aorta unless the adventitia is stimulated, because the aorta has few muscular fibers. The adventitia of the aorta like that of other arteries is very sensitive to painful stimuli, mechanical or chemical. The same is true of the coronary arteries, according to Singer. The conclusion may be drawn that the pain, which is the most striking symptom of angina pectoris, is caused in some cases by an aortalgia (Albutt, Schmidt and Wenckebach) and in others, by a coronarialgia.

The pain originating in the aorta is conducted to the cord by pathways similar to those which Langley demonstrated for pain originating in the heart. Bilateral vagotomy above the depressor nerve and also bilateral extirpation of the superior cervical ganglion did not prevent this pain. Sometimes it is sufficient to extirpate the left stellate ganglion, but usually bilateral extirpation is necessary to abolish the aortic pain.

The painful impulses passing the stellate ganglion reach the cord by the posterior root from the eighth cervical to the fourth thoracic (experiments in collaboration with Dr. R. Singer). After section of these posterior roots, pain reactions from stimulation of the heart or the aorta could not be observed even if the anterior roots were intact. If one wishes to interrupt pain conduction from the heart or aorta without injury to the accelerator nerves, one must cut the posterior roots mentioned. Most of the dorsal root cells that receive pain impulses from the heart and aorta send their axons into the cord independently from the ganglion cells receiving centripetal impulses from the skin and muscles. Even if degenerative changes are produced in the latter group of ganglion cells, by section of the first four intercostal nerves of the brachioplexus one can still produce pain reactions from the heart, the aorta or the stellate ganglion (experiments in collaboration with Dr. Hashimoto).

Centripetal impulses originating in the femoral arteries, as well as those passing along the pelvic, the splanchnic and the cardiac nerves, are conducted in dogs and cats by crossed and uncrossed fibers running in the ventrolateral periphery of the cord (Spiegel and Bernis). There exist not only long tracts conducting visceral pain, but also short tracts that form chains of neurons. This is shown by the fact that stimulation of the centripetal fibers of the pelvic nerves, after a bilateral hemisection of the cord at different levels, for instance in the lumbar segments on the right and the cervical segments on the left, still produces pain reactions (experiments in collaboration with Dr. Abuel).

DISCUSSION

DR. JAMES WHITE: This communication seems extremely important to me from a surgical point of view. We have been getting good results in relieving the pain of angina pectoris by alcohol block of the upper four or five thoracic rami. We prefer this to operative section of the rami, because it is less dangerous. However, occasionally some rami are missed and then it is impossible to operate on account of adhesions. If a section of the posterior roots is equally effective, it might be employed in such cases, and we could then first try the alcoholic injections in all cases. In patients with causalgia after vascular wounds, the pain

is often not relieved by root section, but may be relieved by rami section. Has Dr. Spiegel any information how these fibers run?

DR. E. W. TAYLOR: Is there any physical explanation of the sensitivity of the dura to stretching although it is insensitive to cutting?

DR. T. J. PUTNAM: Has Dr. Spiegel found any local anesthetics which are effective for vascular pain?

DR. E. A. SPIEGEL: In regard to Dr. White's injections, I am surprised that it is not dangerous to destroy the entire root ganglion. It contains efferent fibers running to the heart as well as sensory fibers and, especially in the presence of cardiac disease, I should think that disturbances might result. In regard to the posterior roots, Dr. Denk of Vienna sectioned the first four posterior roots in a patient with pain from syphilitic aortitis. This relieved the pain as far as the fourth segment, but the patient continued to have the discomfort below. I believe, however, that the operation should be undertaken only when the myocardium is in good condition. I cannot believe that sensory impulses are ever transmitted through the anterior root. It is impossible to obtain an action current from the posterior root by stimulation of the anterior. Doubtless, pain is transmitted over many more nerve roots than we ordinarily understand, perhaps spreading from segment to segment by the periarterial plexus. The sensation in the dura is probably limited to cerebral vessels which are stimulated by stretching but not by cutting. In experiments on animals, arterial pain may be arrested by infiltrating with procaine hydrochloride.

CEREBROSPINAL FLUID SUGAR AND THE "STREPTOCOCCUS OF POLIOMYELITIS." DR. FRANK FREMONT-SMITH and MARY ELIZABETH DAILEY.

In a series of publications (*J. Infect. Dis.* **22**:313, 1918; *Am. J. Dis. Child.* **33**:27 [Jan.] 1927; *J. A. M. A.* **91**:1594 [Nov. 24] 1928; *J. A. M. A.* **94**:777 [March 15] 1930), E. C. Rosenow and his associates presented evidence that acute anterior poliomyelitis is caused by a streptococcus. This organism has been cultivated from the throats of patients with this disease, from the spinal fluids of patients in the acute stage of the disease and from the spinal fluids of monkeys in the acute stage of experimental poliomyelitis produced by intracerebral inoculation of glycerolated virus. In the latest publication, photomicrographs are shown of diplococci recovered by smears made from centrifugated cerebrospinal fluid in human cases of acute anterior poliomyelitis. A serum has been prepared, and a reduction in mortality has been reported following its use.

P. K. Olitsky, C. P. Rhoads and P. H. Long (*J. A. M. A.* **92**:1725 [May 25] 1929) were unable to recover streptococci by smear or culture from the spinal fluids of seventeen monkeys ill with poliomyelitis. These authors called attention to the fact that the yeast medium used by Rosenow contains streptococci.

If a streptococcus is present in the cerebrospinal fluid in acute cases of poliomyelitis, one might expect the cerebrospinal fluid to resemble that found in cases of streptococcus meningitis; for example, the sugar content of the spinal fluid should be low, provided this streptococcus ferments dextrose.

In a series of one hundred nonmeningitic patients, fasting nine hours or more, with only minor deviations from the normal cell and protein content, the spinal fluid sugar ranged from 45 to 93 mg. per hundred cubic centimeters. In 94 per cent of the cases the values were between 50 and 75 mg. In a series of seventy-one cases of acute anterior poliomyelitis the spinal fluid sugar ranged from 51 to 97 mg., 90 per cent being between 51 and 75.

It is evident, therefore, that the sugar content of the cerebrospinal fluid is normal in acute anterior poliomyelitis. If the streptococcus is present, it does not appreciably lower the sugar content of the cerebrospinal fluid.

The normal sugar content of the spinal fluid in poliomyelitis might be accounted for by either of the following possibilities: (1) this streptococcus for some reason did not have glycolytic properties in the cerebrospinal fluid; (2) its growth in the cerebrospinal fluid was for some reason so inhibited that no glycolysis occurred.

Were this the case, it might account for the fact that the organism is so difficult to recover and grow from the spinal fluid in human cases of poliomyelitis, and that both the clinical and the pathologic picture are so utterly different from known streptococcus infections of the meninges.

Through the courtesy of Dr. C. P. Rhoads at the Rockefeller Institute, New York, we were able to test these possibilities as follows: On Jan. 8, 1929, Dr. Rhoads received from Dr. Rosenow a culture of streptococcus from a case of poliomyelitis. A transplant from this culture was forwarded to us. Nine tubes of sterile spinal fluid were inoculated directly from this culture and seven other tubes were inoculated with 0.05 cc. of a forty-eight hour broth culture of the streptococcus. These tubes were incubated at 37 C. for from twenty to forty-five hours. In every case but one, a profuse growth of the streptococci took place, such that the formerly clear cerebrospinal fluid became turbid. In twelve instances subcultures were made at the end of incubation. From ten of these the green-producing streptococcus was recovered in pure culture. The other two, each incubated for forty-four hours, were sterile, although earlier growth had obviously taken place. One tube incubated for twenty-four hours remained clear, and showed

Cerebrospinal Fluid Sugar After Inoculation and Incubation

Case No.	Original Sugar mg. per 100 cc.	Incubation at 37 C., Hours	Sugar mg. per 100 cc. Control	Sugar mg. per 100 cc. Inoculated	
1	75	20	72	15	Spinal fluid protein 34 mg. per 100 cc.; cells, 5
		44	62	15	Diagnosis: Chronic epidemic encephalitis
2	70	20	69	9	Spinal fluid protein 28 mg. per 100 cc.; cells, 13
		44	69	8	Diagnosis: Multiple sclerosis
3	60	20	60	11	Spinal fluid protein 26 mg. per 100 cc.; cells, 3
					Diagnosis: Gonococcal urethritis
4	63	24	67	21	Spinal fluid protein 78 mg. per 100 cc.; cells, 12
					Diagnosis: Central nervous system syphilis
5	78	23	78	23	Spinal fluid protein 43 mg. per 100 cc.; cells, 5
		23	80	23	Diagnosis: Old central nervous system syphilis
6	73	21	72	11	Spinal fluid protein 25 mg. per 100 cc.; cells, 2
		45	70	15	Diagnosis: Hysteria
		45		18	
7	92	24	90	91	Spinal fluid protein 28 mg. per 100 cc.; cells, 0
					Diagnosis: Progressive muscular atrophy
8	73	20	73	12	Spinal fluid protein 22 mg. per 100 cc.; cells, 1
		44	74	10	Diagnosis: Epilepsy
9	72	20	71	26	Spinal fluid protein 26 mg. per 100 cc.; cells, 7
		44	70	18	Diagnosis: Multiple sclerosis

on smear only a few short chains of streptococci. For some reason the organism had not grown in this tube. Thus of sixteen tubes of spinal fluid inoculated, fifteen showed profuse growth. In each of these fifteen tubes the spinal fluid sugar (determined by the method of Folin and Wu) showed marked diminution, the initial sugar before inoculation ranging from 60 to 92 mg. per hundred cubic centimeters, while the sugar after inoculation and incubation ranged from 8 to 26 mg. This is about the sugar value found in acute purulent meningitis. In the one case in which the spinal fluid remained clear the sugar was 92 mg. per hundred cubic centimeters before inoculation and 91 mg. after inoculation and twenty-four hours of incubation.

To show that glycolysis was indeed due to the streptococcus and not to glycolytic ferments already in the cerebrospinal fluid, fifteen control tubes of spinal fluid were incubated for from twenty to forty-five hours at 37 C. In one case only was the sugar value lower after incubation, falling from 75 to 62 mg.; in the other fourteen no appreciable change in the sugar content took place. This confirms the work of A. G. Kelley (*South. M. J.* **16**:407, 1923) and is in direct contradiction to the results of K. Chevassut (*Quart. J. Med.* **21**:91, 1927).

Conclusions.—1. The level of sugar in the spinal fluid in acute anterior poliomyelitis is normal. 2. A strain of streptococcus isolated by Dr. Rosenow from a case of poliomyelitis grew profusely and repeatedly in human cerebrospinal fluid,

and this growth was regularly accompanied by a marked reduction in the sugar content of the cerebrospinal fluid. 3. Incubation of normal sterile cerebrospinal fluid at 37 C. up to forty-five hours does not result in a diminution of the sugar content.

DISCUSSION

DR. D. GREGG: Was the Miss Chevassut to whom you refer the same one who recently had work published on the subject of multiple sclerosis with Dr. Purves Stewart?

DR. D. MACPHERSON: What were the spinal fluid observations in Dr. Rosenow's experimental animals?

DR. FREMONT-SMITH: This is the same Miss Chevassut. Dr. Rosenow did not report any chemical studies of his experimental animals.

NEW YORK NEUROLOGICAL SOCIETY AND THE NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Joint Meeting, May 13, 1930

MOSES KESCHNER, M.D., *Chairman, in the Chair*

CLINICOPATHOLOGIC PRESENTATION FROM THE NEUROLOGIC SERVICE OF MOUNT SINAI HOSPITAL

PERICAPILLARY ENCEPHALORRHAGIA FOLLOWING THE ADMINISTRATION OF ARSPHENAMINE; FATAL ISSUE; NECROPSY OBSERVATIONS. DR. ISRAEL STRAUSS and DR. JOSEPH H. GLOBUS.

Clinical History.—B. G., a woman, aged 25, was admitted to Mount Sinai Hospital on Dec. 5, 1929, in a state of deep coma. The patient's mother was unable to give reliable data beyond the fact that, as far as she knew, the patient had never been seriously sick until the onset of the present illness, and that on the day preceding admission, about 7 p. m., the patient told her that she was going to a physician to be treated for a "cold." She returned half an hour later, very pale and extremely nauseated. She vomited repeatedly that night, and complained of severe headache. She went to bed. On the next day, the headache became more persistent and she vomited several times. At 4 p. m. on the day of admission she suddenly became irrational and complained of a more severe headache. Soon after, she had a generalized convulsion and passed into deep coma.

It was subsequently learned from the attending physician that the patient had had a primary syphilitic infection of the vagina several months previously, and that several days before the onset of the recent illness she had received a second or third injection of arsphenamine.

Examination.—The patient was in deep coma. There were: gross nystagmoid movements of the eyes; left central facial paralysis with deviation of the tongue to the left; deep reflexes hyperactive (the left more so than the right); transient bilateral ankle clonus; absent abdominal reflexes, and submaxillary and mandibular adenopathy. There was no albumin, sugar or microscopic abnormality in the urine. The Wassermann test of the blood gave a 4 plus reaction. The cerebrospinal fluid was clear and contained three cells. The colloidal gold curve was 0112232100, and the Wassermann tests of the cerebrospinal fluid read as follows: negative with 0.1 and 0.2 cc.; positive with 0.4 and 0.6 cc. (++++), and also with 0.8 and 1.0 cc. (++++).

Course.—On the day of admission, the patient had another generalized convulsion which at first had the character of a jacksonian seizure. It began with twitchings of the fingers of the left hand and spread rapidly to involve the entire

body. Despite active therapy, including the intravenous administration of sodium thiosulphate, she declined rapdily and died on December 7, two days after admission.

Necropsy.—Gross Anatomy: The brain was voluminous and massive and showed moderate flattening of the convolutions. The pial vessels were markedly congested. The orbital surface of the right cerebral hemisphere showed marked discoloration due to the subpial extravasation of blood. On sectioning, the most striking alteration was seen in both optic thalami, the centers of which were occupied by areas of discoloration due to the diffusion of extravasated blood. Similar areas of discoloration were seen in the cornu ammonis, in the tegmentum of the midbrain and in several areas of the pons and the cerebellar cortex.

Microscopic Anatomy: The striking alterations were: (1) pericapillary hemorrhages, (2) moderate gliosis about the hemorrhagic areas and (3) moderate demyelinization about the hemorrhagic zones. The interpretation of the histologic alterations will be given in a full report of this case to appear elsewhere.

DISCUSSION

DR. LOUIS CASAMAJOR: How much arsphenamine did this patient receive, and in what form? With the large amount of arsphenamine being used at the present, I am surprised that cases like this do not occur more often; maybe they do. Patients often receive an overdose of arsphenamine, and although I have seen toxic manifestations similar to a typical neuritis at times, with changes in the liver, I have never encountered a picture as remarkable as this. When the first sections were shown on the screen, I wondered why the perivascular hemorrhages were not those of epidemic encephalitis. Dr. Globus answered the question by pointing out that the perivascular hemorrhage in encephalitis occurs around the larger arterioles, whereas here it is purely from the capillaries. Is there any known reason why those particular blood vessels should have been thus affected? How frequently does this occur, and is there anything that would put one on guard against giving arsphenamine in certain cases?

DR. MICHAEL OSNATO: What was the condition of the large vessels in this case? One knows what may happen in syphilitic aortitis following the ill advised use of arsenicals with the occasional precipitation of disastrous results. It occurred to me that in this case there might be a syphilitic disease of the small blood vessels analogous to that which occurs in the large vessels about the heart, and that this condition was aggravated by the arsenical.

Was there any indication of syphilitic arterial disease in the large vessels?

DR. MOSES KESCHNER: What does Dr. Globus think of the recent paper by Eugen Pollak that I showed him, which concerns a study of four cases of injury to the brain by arsphenamine. Pollak stated that he has observed that arsphenamine can affect the brain in four different ways: (1) with a picture similar to that shown in the case presented here, with involvement of the capillaries, which he calls the purpuric type; (2) one in which the arsphenamine seems to affect only the glia of the brain and does not affect the vessels; (3) another is a combination of the first two and (4) a type, which he designates as the Herxheimer reaction of the brain, not like the angioneurotic edema observed in the skin and mucous membranes, but a productive infiltration of the mesodermal elements of the brain. Pollak also emphasized that none of the four types showed syphilitic arterial disease to which pathologists could attribute the cerebral changes.

DR. ISRAEL STRAUSS: I do not know what dose of arsphenamine was used. Examination of the other organs of the body gave negative results. There was a curious feature about this case, even pathologically, to which I called Dr. Globus' attention. My first impression of the brain was that I should find the so-called hemorrhagic encephalitis; I expected to find hemorrhages scattered throughout the brain. The remarkable thing about this case, which Dr. Globus and I cannot understand, is the fact that the hemorrhages really are localized in so few and such small areas of the brain. Dr. Globus stated that small areas

of the brain were involved—the corpus striatum, the pons and the midbrain. Why are just those few areas in the brain selected as the site of injury? During the early use of arsphenamine, a great many cases of so-called hemorrhagic encephalitis were reported. I believe that one of the reasons why this serious complication is not observed today is that arsphenamine is not given without the preparatory administration of mercury or bismuth. Arsphenamine is a toxic drug. Unquestionably some persons are more susceptible to it than others. One does not know how the individual patient will react to the drug. Fortunately, it is rare to observe such a serious complication as the one reported here, and I think that one of the reasons is that preliminary medication is used before giving arsphenamine, especially soon after the primary lesion.

Dr. Joseph H. Globus: The large vessels were uninvolved, and the most striking thing about the case was that the capillaries were the only structures involved. Even the small pericapillaries were intact, and the reason is that the capillaries are terminal vessels and the heavier metals have an affinity for them. It is because of this affinity for the heavier metals that the blood vessels in the brain are so frequently involved, as in some of the preparations here.

Experience with one case does not entitle me to regard myself as an expert in encephalitis caused by arsphenamine. This may be a quantitative reaction. With a substance such as arsphenamine, there may be a rupture of the blood vessels with exudation of blood followed by absorption in the tissues. With a smaller quantity, or a less noxious preparation, there would be a more mild reaction, and the material might escape without actual rupture of blood vessels and without the extravasation of blood. I think that Pollak was dealing with quantitative reactions rather than with true qualitative differences.

Invasion of the Brain by Parasitic Cysticercus; Fatal Issue; Necropsy.

Dr. Israel Strauss and Dr. Joseph H. Globus.

Clinical History.—J. K., a Russian Jew, aged 30, a salesman, was admitted to Mount Sinai Hospital on Oct. 29, 1929, complaining of headache, vertigo, vomiting and a "drumming sensation" in the left ear. He gave a vague history of a venereal infection in 1926. In August, 1927, he began to receive deep roentgen therapy for enlarged cervical glands, which were regarded as tuberculous. He received four courses of roentgen therapy. After the last treatment, about ten days prior to admission, the patient began to experience an occasional mild headache; several days later, the headache became more intense and there was repeated vomiting. The headache, occipital in character, was accompanied by a drumming sensation in the left ear and by marked vertigo. The patient then went to bed. During the last few days, obstinate constipation had developed and for sixteen hours before coming to the hospital he was unable to micturate.

Examination.—The patient was acutely ill. He was afebrile; the pulse rate was slow. A mass was felt at the left angle of the mandible. There was rigidity of the neck with a bilateral Kernig sign. The deep reflexes were depressed. There were no pathologic reflexes. The cranial nerves were intact except for a suspicious blurring of the margins of the disks. A lumbar puncture yielded cerebrospinal fluid under moderately increased pressure, which at first was somewhat bloody but became successively clearer. Another lumbar puncture yielded pinkish cerebrospinal fluid, under increased pressure, which contained many red blood cells. No organisms were found in the smears. Roentgenograms of the skull and chest were normal.

Course.—For a few days the patient felt better, but there was apparent progression. The blurring of the margins of the disks became more pronounced and a right facial weakness and a left Babinski sign appeared. The papilledema continued to increase; retinal exudates and hemorrhages appeared.

On the eleventh day in the hospital, following a sudden exertion, the patient had a convulsive seizure. He lost consciousness. The eyes and mouth were drawn to the left and spasticity developed in the left upper extremity. The

signs in the pyramidal tract on the left side became more pronounced. An expanding lesion in the right hemisphere was diagnosed. A ventricular estimation with injection of air was advised. On November 12, trephining over both occipital horns was performed. On the right side, at a depth of 2 cm., tarry blood was obtained. At a depth of 5 cm., the ventricle was entered and a very small amount of blood-tinged cerebrospinal fluid emerged. Aspiration of the left horn was attempted, but no fluid was obtained. Following the ventricular puncture, the patient's condition grew progressively worse and he died on the seventeenth day in the hospital, twenty-seven days after the onset of symptoms.

Necropsy.—Gross Anatomy: There were several circumscribed greenish areas over the surface of the brain. One was over the region of the left middle frontal convolution, about 0.5 cm. in diameter, with a zone of softened brain substance surrounding it. Another was situated in the right frontal lobe and was of about the same size and appearance. Still another, on the inferior surface of the left cerebellar hemisphere, presented a small hemorrhagic discoloration, about 2 mm. in diameter. In the right occipital lobe, near the mesial surface, there was a disorganized area of brain substance, which measured about 1 cm. in diameter and corresponded to a recent operative puncture. Along some of the larger vessels on the surface of the brain there was a slight amount of opacity of the leptomeninges. The base of the brain showed no abnormality. On sectioning the brain, numerous hemorrhagic focci were found throughout the cerebral hemispheres and the cerebellum. They varied in size from about 3 mm. to about 2 cm. in diameter, with all gradations between. They were either oval or circular. All showed grayish centers. They were well demarcated from the surrounding tissues of the brain with no transition of softened areas except in the large foci. The distribution was very irregular. They were found in both hemispheres and were about equally numerous in each. They were situated either in the cortex, directly underneath the surface, or close to the ventricular cavity, within the white substance.

Microscopic Anatomy: Sections through several hemorrhagic foci of the brain displayed solid masses of well isolated collections of elements of large mononuclear cells, containing many giant cells. The majority of the cells were polygonal and rich in protoplasm and contained deep-staining nuclei. The cellular masses were surrounded by wide zones of extravasated blood. The circumscribed collections of cellular structures gave the impression of an inflammatory process, a reaction to parasitic invasion. A small fragment of tissue had been previously removed by the surgeon when the ventriculography was performed. In this, material structures were noted, which were highly suggestive of hooklets, scolex and a parasitic membrane. This case is now being prepared for publication in a more extensive form; it will be illustrated by photomicrographs of the unusual histologic features.

DISCUSSION

Dr. Lewis D. Stevenson: These cases seem to be rare, and this is an excellent demonstration of parasitic invasion of the brain. At Bellevue Hospital, we have encountered only one cysticercus of the fourth ventricle; there was no diffuse infiltration as in this case, and there were no hemorrhagic areas.

Dr. Moses Keschner: It is of interest to note that there was scarcely any eosinophilia. Numerous routine examinations of the blood were made, and at no time was there more than 1 per cent of eosinophils in the blood.

Tumor of the Craniopharyngeal Duct (Cyst of Rathke's Pouch), with Signs of the Picture of Basal Meningitis; Fatal Issue; Necropsy. Dr. Israel Strauss and Dr. Joseph H. Globus.

Clinical History.—P. Z., a boy, aged 10, was admitted to Mount Sinai Hospital on Feb. 1, 1930. Two months previously a severe frontal headache had developed. A diagnosis of sinusitis was made. Shortly after, the patient improved temporarily; however, he remained somewhat dull and sluggish, and one week

before admission began to complain of frontal headache, vomited frequently and became drowsy.

Examination.—The patient was poorly developed, dull, apathetic and indifferent. There was pallor of the temporal side of the right optic disk, and diffuse pallor of the left disk. There was some weakness in the left external rectus, a right facial paresis of central type, a deviation of the tongue to the right and moderate weakness in the right upper and lower extremities. The deep reflexes were more active on the right; the abdominal reflexes were not obtained. A Babinski sign was present on the right side. Moderate Kernig and Brudzinski signs were elicited. There were choreo-athetoid movements of the left upper extremity and increased tonus in both upper extremities, with a suggestion of cogwheel phenomena on the left side. The gait was staggering, with a tendency to fall backward. The head was tilted to the left. There was limitation of the visual fields, suggesting a right homonymous hemianopia. Examinations of the blood and the urine gave negative results. The cerebrospinal fluid contained 480 cells, with 95 per cent mononuclear elements. A second lumbar puncture showed 70 cells, with 90 per cent mononuclear cells. No organisms were found in the cerebrospinal fluid.

Course.—The patient's condition varied from time to time. Periods of extreme restlessness or somnolence would alternate with periods in which he was fairly alert, observant and talkative. One night, soon after admission, the pulse became feeble and the breathing shallow. Throughout the clinical course the neurologic signs varied in intensity, but the type did not change. The most persistent signs were those of meningeal irritation, which gradually increased in severity. A right hemiplegia also appeared. The patient's temperature never rose above normal during his stay in the hospital. The Pirquet and Mantoux tests gave negative results. A ventricular estimation was made which indicated that the lateral ventricle on the left side was dilated. No other information as to the character and location of the lesion was obtained. Following the operation the child grew worse. Transfusion was performed but he did not rally. He died on March 3.

Necropsy.—Gross Anatomy: The brain was voluminous and unusually soft and showed evidence of increased intracranial pressure. On lifting the frontal lobes, some difficulty was encountered due to an adherent tumor mass in the region which was greatly widened, while the upper part of the tumor occupied the interpeduncular space and was partly embedded in the base of the brain. The tumor measured about 5 cm. in longitudinal and 3 cm. in transverse diameter. It was generally firm, with cyst formation in some areas. It was pinkish white and was surrounded by a firm capsule. The optic nerves were much flattened and compressed by the lateral parts of the tumor mass. The interpeduncular cistern posterior to the tumor was tremendously dilated with a dark opaque material. The pituitary gland could not be found after the extirpation of the tumor and was assumed to form part of the tumor mass. On section of the brain in a median sagittal plane, the tumor was found to occupy completely the third ventricle. It shelled out easily from the ventricular cavity and on section showed a granular appearance, a rather glistening surface and an occasional small cyst. At the anterior part, on its under surface, a small nodule measuring about 3 mm. in long diameter, wedge-shaped and attached to the capsule of the main tumor was found, and since no pituitary gland was found in the sella turcica it is probable that the small mass was all that was left of the pituitary gland. The lateral ventricles were symmetrically enlarged.

Microscopic Anatomy: The histologic picture of the tumor was that of a neoplasm of the craniopharyngeal duct (cyst of Rathke's pouch).

Comment.—The meningeal signs in this case dominated the clinical picture; when the high cell count in the cerebrospinal fluid is added to the manifestation of meningeal irritation it becomes obvious why a diagnosis of tuberculous meningitis was favored. The pallor of the disks was not characteristic of a postneuritic atrophy, and could also be a part of the clinical picture of a basal meningitis accompanied by internal hydrocephalus.

In the light of recent publications (Beckmann, J. W., and Kubie, L. S.: A Clinical Study of Twenty-One Cases of Tumour of the Hypophyseal Stalk, Brain **52**:127 [July] 1929) on the tumors of the Rathke pouch, pleocytosis and manifestations of meningeal irritation need not be considered as objections to a diagnosis of a suprasellar neoplasm.

PINEALOMA IN A CHILD, AGED 20 MONTHS; FATAL ISSUE; NECROPSY. DR. ISRAEL STRAUSS and DR. JOSEPH H. GLOBUS.

Clinical History.—M. S., a boy, aged 20 months, was born at term, with normal delivery. Development had been normal until the onset of the present illness. Five weeks before admission to Mount Sinai Hospital, the child fell, striking the back of his head. There were no symptoms until three hours later, when he had nose bleed that lasted about ten minutes. Two weeks later, he began to vomit everything taken by mouth, immediately after ingestion. During that time, he passed through a brief episode, of ten minutes, in which the "whole body" became rigid. He was taken to a hospital where he remained for two weeks. There was no improvement and as no definite diagnosis was offered the mother took the child home. He continued to vomit everything he ate and became apathetic and more obviously ill. On the day before admission to Mount Sinai Hospital, the patient had another tonic spasm, which lasted about ten minutes and recurred on the following day. This time the child lost consciousness and remained in a rigid state until he was brought to the hospital four hours later.

Examination.—The child was in a state of coma. The body was rigid with all extremities in extension. The attitude was suggestive of decerebrate rigidity. The knee reflexes and abdominal reflexes were absent. The disks revealed questionable papilledema. A lumbar puncture yielded clear cerebrospinal fluid, under markedly increased pressure, containing 60 lympocytes per cubic millimeter. The Wassermann test, colloidal gold curve and cultures of the cerebrospinal fluid gave negative results. The patient died on the day of admission.

Necropsy.—Gross Anatomy: The convolutions of the brain were flattened and gyri shallow. There was marked internal hydrocephalus. On lifting the occipital lobes, a soft, pinkish, necrotizing tumor mass was exposed. This tumor mass could be traced upward to the pineal body and was found to extend downward to the vermis of the cerebellum, to which it was intimately attached. The pineal body was considerably enlarged. On section of the brain in a median sagittal plane, the tumor was found over the quadrigeminate plate, extending backward and invading the anterior portion of the vermis of the cerebellum. The tumor showed a varied appearance. At the anterior extremity it was granular, soft and necrotic. In the most dorsal portion and somewhat posteriorly it consisted of a large nodule of hard granular material. More ventrally and posteriorly, where it invaded the cerebellum, the tumor was smooth. Another nodule was found invading the middle surface of the right occipital lobe in the region of the common stem of the parieto-occipital and calcarine fissures. It measured here about 1 cm. in diameter. In the region of the habenula a small conical structure, about 2 mm. in size, was found; it was the pineal gland. The third ventricle and lateral ventricles were markedly and symmetrically dilated.

Microscopic Anatomy: Sections of the tumor revealed the characteristic structure of a pinealoma, and in certain areas bore a striking resemblance to the architecture of the pineal gland, which was found as an independent structure in the case. More detailed description of the anatomic features in this case will appear in a later issue of the ARCHIVES.

NIEMANN-PICK'S DISEASE; FATAL ISSUE; NECROPSY. DR. ISRAEL STRAUSS and DR. JOSEPH H. GLOBUS.

Clinical History.—S. H., a girl, aged 10 months, was admitted to the pediatric service of Mount Sinai Hospital on April 15, 1929. She was born at

term; delivery was normal; she weighed 4¾ pounds (2.1 Kg.) at birth. She was breast fed up to 7 months when weaning was begun, but feeding the child by bottle was difficult and from about three to five days after she had been weaned she began to vomit. The vomiting was not definitely related to meals and was projectile, consisting of food previously taken. About six weeks before admission, she began to cough, and the temperature was elevated, ranging from 101 to 105 F. During the three weeks prior to admission, the child had acted peculiarly. She would open her mouth, place her fingers in it and pull down the lower jaw as though she were in pain. She became less active and less interested in her surroundings.

Examination.—The patient was marantic; the head appeared large, the fontanels were open and the scleras were blue. The skin had a brownish tinge and presented a general miliaria. The liver and spleen were enlarged. The fundi revealed large "cherry red" spots.

Course.—In view of the observations of the ocular fundi, the marked enlargement of the liver and spleen and the unusual number of lymphocytes in the blood showing vacuolization, a diagnosis of Niemann-Pick's disease was suggested. There were varied opinions in the interpretation of the retinal observations, but it was generally conceded that the fundi were sufficiently typical to warrant the diagnosis. Splenic tissue was removed by puncture and was reported as presenting features of Niemann-Pick's disease. During the child's stay in the hospital, there were several transient elevations of temperature, some reaching 104 F. The laboratory data of significance were: hemoglobin 68 per cent; red blood corpuscles 4,250,000; white blood cells 16,000, with 51 per cent polymorphonuclear leukocytes and 49 per cent lymphocytes, of which 75 per cent had a vacuolated cytoplasm. The blood cholesterol was 290 mg., and the total blood fat, 1.43 Gm. Urinalysis gave negative results, except for double refractile bodies under polarization.

The child was allowed to return home, but she was readmitted on September 5, because of the sudden appearance of fever and anorexia. She had lapsed into deep coma and was brought to the hospital in this condition. Examination showed a pneumonic infiltration in the right lung. The temperature was 105 F. The child died soon after admission.

Necropsy.—Gross Anatomy: The brain was small; it was rubbery. The meninges were dull. The general appearance was typical of the brain in amaurotic family idiocy.

Microscopic Anatomy: Study of numerous sections revealed a widespread degeneration of the nerve cells throughout the central nervous system. It was characterized by swelling of the cells with displacement and pyknosis of the nuclei, granular degeneration of the cytoplasm and swelling of the cell processes. The blood vessels showed no evidence of an inflammatory reaction.

DISCUSSION

DR. ISADORE GOLDSTEIN: In Niemann-Pick's disease the cherry red spot occurs as in amaurotic familial idiocy. The spot will take the shape of the normal fovea. The edema is interesting; there are changes in the choroid, the ganglion cells and the ganglion cells accompanying the nerves in the posterior part of the eye. There is an anatomic change in Niemann-Pick's disease which differs from that of amaurotic family idiocy. These will be described in a future communication. The fundus picture is practically the same in the two conditions.

DR. SAMUEL BROCK: Have the splenic and liver changes of the Niemann-Pick syndrome ever been described in connection with the juvenile Spielmeyer type of amaurotic idiocy?

DR. JOSEPH H. GLOBUS: In none of the five cases of the juvenile form of amaurotic familial idiocy, the so-called Spielmeyer-Vogt type, were changes noted in the spleen or other organs.

SIMMOND'S DISEASE; FATAL ISSUE; NECROSPY. DR. ISRAEL STRAUSS and DR. JOSEPH H. GLOBUS.

Clinical History.—J. S., a woman, aged 53, was admitted to Mount Sinai Hospital on Oct. 26, 1929, complaining of wasting and general weakness. One year previously she began to show evidence of lowered resistance. She became subject to repeated colds, developed a persistent cough and lost weight progressively. The loss of weight and the sapping of strength advanced rapidly. Three weeks prior to admission she had a gastro-intestinal upset with diarrhea, moderate elevation of temperature and cramps, which lasted about a week.

Examination.—There was marked emaciation with almost complete loss of all the subcutaneous fat tissue. The patient weighed only about 70 pounds (31.8 Kg.) (normal weight 110 pounds [49.9 Kg.]). There was no icterus. The blood pressure was 125 systolic and 80 diastolic. The hemoglobin was 58 per cent, with 3,000,000 red blood corpuscles. The Rehfus test showed complete achlorhydria in spite of 0.5 mg. of histamine. The gastric ferments were present. Roentgenologic examinations showed that the chest and the gastro-intestinal tract were normal. The blood chemistry was normal.

Diagnosis.—In spite of the great loss of weight, the achlorhydria and the anorexia, the clinical picture was not considered to be that of malignancy. The only explanation offered for the extreme emaciation was the possibility that the case was an instance of Simmond's disease.

Necropsy.—Gross Anatomy: Aside from mild atherosclerosis of the vessels at the base of the brain and some dulling of the leptomeninges, the brain was normal. There was a peculiar yellowish-tan hue to the surface of the brain. The same écru color was seen on the cut surface of the brain.

Microscopic Anatomy: The pituitary gland showed alterations typical of Simmond's disease: marked fibrosis of interstitial tissue and a reduction in the glandular elements.

DISCUSSION

DR. SAMUEL BROCK: Were there changes in the suprarenals, thyroid, ovaries and other ductless glands in this case? I ask because Simmond's syndrome is practically identical with that described by Claude and Gougerot years ago—an acute polyglandular insufficiency with a fatal termination. Because of this great resemblance, one wonders whether it is correct to attribute all these phenomena to disease of the pituitary glands.

DR. JOSEPH H. GLOBUS: The other glands were examined and revealed no changes.

Book Reviews

REVISION DE LA DOCTRINE DES LOCALISATIONS CÉRÉBRALES. By R. BRUGIA. Price, 24 francs. Pp. 190. Paris: Masson et Cie, 1929.

As Professor Pierre Marie states in the preface to this book, "It comes timely and marks a new orientation." Chapter I, Theory of Centers: The author reviews the localization of different functions in definite areas of the brain. He points out the influence of certain antiquated ideas on the tendency to localize the nervous functions in anatomic centers and qualifies this tendency as a result of merely a mental attitude rather than of critical scientific thought. The experience with cerebral traumas during the world war discredited the belief in the existence of cortical centers as agglomerations of nerve cells possessing the quality to generate the functions attributed to them. Giving a brief résumé of the "neuron theory" as a basis for the assumption that each neuron is a self-sufficient anatomic unit, a reservoir of forces under tension, which like a "fire in the furnace flares when poked," the author shows how from this theory the accepted scheme of the simple reflex arc was formed (afferent conduction, excitation of the ganglion cell, efferent conduction) and how in further elaboration of this theory the nerve cell bodies become invested with specific function to transform the external impressions into reflexes. To the "neuron theory" of Ramon y Cajal, the author opposes the "neurite" theory of Apáthy and refers to the works of Loeb, Bethe, Donagio, Bielschowsky, Held and Pierre Marie.

Chapter II, Segmental Theory and the Neuromyon: To the doctrine of centers, according to which two types of neurons (afferent and efferent) form the double corticospinal pathway (ascending and descending), the author opposes the segmental or metameric theory. According to the latter, the whole nervous system is regarded as a colony or a series of units (metameres). Each metamere is provided with a pair of nerves (neuromere), one sensory (ganglionic), derived from the respective dermatomere, the other motor, ending in the myomere. There are no centers, every reaction being integrated by the totality of the metamere and without specific intervention of cellular elements. The cell bodies take part in the conduction of a nerve impulse, but do not generate it; they probably serve as accumulators and distributors of energy in a potential state, but their main function is trophic. Cerebral, as well as spinal, metameres exist. The cerebral hemispheres are nothing but the appendixes of the metameric nervous system. There are as many of the superimposed reflex arcs carrying higher neuropsychic functions as there are myomeres. The diversity of spinal and of cerebral metameric reactions is due only to the specific character of receptor surfaces and to the type of instruments of reaction. The whole anatomicophysiologic complex is the specific and essential element of any nervous or psychic phenomenon. This complex is what Patrizi called the neuromyon. Sensation and movement constitute the first and the last stage of thought. However, the wave of the nerve impulse does not exhaust itself in the kinetic effect; it continues its course through the sympathetic system; it reflects on the "milieu intérieur" and on its way provokes the reactions on the part of endocrine glands. There are kinetic, volitional, emotional neuromyons and within them the exteroceptive, interoceptive, general and local, simple and associative, primary and reproduced reactions intermix, alternate, associate with or elude each other.

Chapter III, Simple Neuromyonic Reactions (Spinal Reflexes): The most elementary form of nervous activity is the reflex. Wundt considered simple reflexes as remnants of the will outdated by evolution. Herring considered them as fossilized intelligence. In certain reactions of lower animals (insects), such as flying toward a flame, and also reactions of plants, such as turning to the insolated

side, Loeb recognized the essence of tropism. This orientation of the body in space is always one and the same thing in animals provided with a nervous system as in plants without any nervous system. This fact excludes that such reactions depend on the central nervous system. With further development and adaptation of the protoplasm, the nervous system becomes differentiated, and with it the tropism rises to an ever higher dignity and importance. The elementary reflex, as compared with psychic phenomena, is what an ameba is to the more developed forms of the animal kingdom. The ameba is a primitive structure; the elementary reflex is the simplest mechanism of life. However, this mechanism is not created by the central activity; in other words, it is not determined as the result of the work of specific nerve cells. The spinal and also the cerebral neuromyons are not provided with a double pathway so that one would carry the material from the environment into an internal laboratory, while the other would export this material transformed into impulsions. On the contrary, the afferent and the efferent fibers both represent the sections of one and the same pathway united by a protoplasmic bridge of junction which is the presumed ganglionic center. The law which rules is always the law of the anterograde movement of the stimulation. The tropism precedes (antedates) the sensation, and when the former acquires the aspect of a reflex it becomes a neurotropism and a psychotropism. The functions of the organism are not caused by the nervous system, but are regulated by the latter. The primary physiologic unity (primary integration) is not neurologic but physicochemical. In the course of embryonic evolution the latter, however, rapidly (second month) combines itself with the action of the nervous system, which, by neurologic integration, assures a greater variability of reactions. Thus, fundamental myotonic activity antedates nervous activity; the latter will give to the former perfection, completeness and harmony, but cannot cause it for the simple reason that what follows cannot be the cause of what precedes.

Chapter IV, Neuromyonic Chain-Reactions (Instinctive Reflexes): The author gives a definition of instincts as higher types of reflex reactions. The physiologic nature of the instinct consists of a succession of chain reflexes, a coordinated series of simpler reflexes without any intervention of nerve cells as centers. The instinctive reactions are set into motion not by the action of the cerebral hemispheres but merely with their collaboration. Heredity is at the basis of instincts; heredity transmits only the specific properties of the germ plasm, which, of course, are independent of nerve centers.

Chapter V, Motor Coordination and the Metameric Theory: Locomotion is the play of external stimulations, not the result of central action. There are no centers which compel the muscles to contract in a preestablished order. Metameres are independent and self-sufficient. The problem of spontaneous muscular contraction resides in physics and chemistry. It is the predominance of calcium ions that prevents a skeletal muscle from contracting rhythmically. It is enough to put such muscle into a solution of sodium chloride or sodium bromide (isotonic to the blood serum) to make the muscle recover the property of contracting rhythmically. There is no need of a center for idiomuscular synchronous contraction of the excised heart. The neuraxis of each animal species possesses autonomic metameric mechanisms for locomotion on the ground, in the air and in water. The coordination of visceral movements, such as those of the heart, of respiration, etc., are not caused by the action of centers but are subordinated to the laws of transmission between independent metameric sections of the organism.

In the four following chapters (VI to IX), the author takes to task the concepts of sensorimotor centers, psychic centers, centers of speech and centers of inhibition, and with a remarkable skill of argumentation contests and denies their existence. To speak of cortical cells as instruments of charge and of discharge in the sense that they can reflect on other cells the excitations which they received, is a vain and inconsistent play of words. The symptoms that the classic school attributed to lesions of the temporal convolutions (psychic deafness) are not real

but express damage to adjacent regions, i. e., to the underlying association fibers or to the central gray nuclei. Vision is the only specific sensory activity which has a cortical localization, but this localization is anatomic and not physiologic. In other words, it indicates the passage of fibers and not a central activity inherent to the structural individualization.

Unlike the muscular effect of electric stimulation of the cortex, the muscular reaction to mechanical stimulation of the cortex is in abeyance and to chemical stimulation this reaction develops only after a certain period of time. According to Loeb, cortical excitability is questionable. He thinks that electrization also would remain without effect if the lines of current did not spread down the underlying myelinated fibers. Schafer showed the inverse relationship between the thickness of the cortex and the effect of its electrization. To regard cortical excitation as a kind of "furnace fire which flares when poked" is an absurdity. Motor palsies produced by lesions limited exclusively to the cortex rapidly improve and can completely clear up. After the destruction of small cortical areas, the innervation can be reestablished through the collateral pathways in a manner analogous to the collateral circulation establishing itself following the obstruction of the blood vessel. Mutilation of the cortex does not affect mental capacity but does affect execution. Perception is not the result of the activity of centers, but is due to the association ability of the pallium. One cannot regard the motor area of the cortex as the center of voluntary impulses. The concept according to which different cortical spots, believed to be psychomotor centers, correspond to the apparatuses receiving the sensation from the periphery and transforming them into voluntary impulses is absurd. The same is true with regard to the so-called psychic centers. The belief in the existence of centers of association is as anthropomorphic as is the invention of centers of coordination. Neuropsychic phenomena are nothing but associations between one or several reactions in a state of activity and the residuals of previous reactions. In other words, they are cerebral reflexes modified by individual experience. (Brugia does not quote the theory of conditioned reflexes but this often comes to the mind of the reader when following the text.) The author criticizes the thesis of Flechsig, according to which the cortex is divided into areas of projection and of association. Not all projection fibers serve to convey effector reactions. The rôle of many of them is to join the cortex with subcortical nuclei, and there is no reason to consider the latter as having no part to play in the intellect.

The argument for the theory of the localization of psychic functions in different areas of the cortex is that a function can depend on structure, lesion of which abolishes this function; but surely a function does not depend on a given structure if only once it has been shown that lesion of this structure left unaltered the function attributed to it. In regard to the functions of the cerebral lobes, this has been the case many a time. The tendency to explain histologically the mechanism of mental processes is as absurd as to try to explain the nature of electric current by studying the fibers of an electric wire. Sensory function belongs to the whole cortex, not to any particular area of it. Neither morphology, clinical observation nor pathologic researches seem to give foundation to the presumed topographic division of the cerebral cortex into areas and territories of varying dimensional extent, each having a different function. Speech is a form of associative reflexes. The work of Pierre Marie undermined the very foundation of the theory of cortical centers of speech. It is the function that creates the organ, not the reverse.

There are no centers of inhibition; inhibition is not an inherent specific property of nerve cells, but the results of many factors, among which chemical factors are important. It is enough to recall to mind the moderating influence of certain secretions (parathyroid, for example) in order to admit the existence of chemical inhibition.

In chapter X the theory of neuromyons is put forward as a substitute for the concept of centers. The author discusses phenomena such as dreams, hallucinations, delusions and delirium.

Chapter XI, Neuromyonic Mechanism of Epilepsy: This is an interesting and salutary chapter. The muscular violence of epilepsy and of epileptic syndromes usually is attributed to an explosion of impulses from the motor cortex of the brain, from the direct stimulation of the latter. The author believes that, on the contrary, the disturbance is produced by a morbid process which acts not as a stimulant of the centers of action, but as a factor abolishing the inhibitory mechanisms, so that nervous reactions follow the shortest way with resulting violence and excess. Against all logic, neuropathology persists in accepting the view that epileptic paroxysms are the result of irritation exalting cortical activity. The author considers such a view absurd, when, for example, the paroxysms occur during sleep or during acute anemia of the brain caused by hemorrhage. There is no irritation which, while acting on the nerve elements, would not injure their chemistry (nutrition) to the detriment, not to the advantage, of their functional potentiality. Mechanical stimulation has no convulsant effect on the cortex unless applied to the white fibers of the centrum ovale; chemical agents applied to the cortex produce convulsant effect not immediately but only after a more or less long period of time has elapsed; the convulsant effect of cortical electrization is in inverse proportion to the thickness of the gray cortical matter and, therefore, one is brought to believe that convulsions caused by electrization of the motor area are due not to the excitation of the cortex but to the diffusion of the current over the corticonucleomedullary pathways. For the creation of epilepsy a specific preparation of the cerebral neuromyons is necessary. There are numerous reasons which bring one to believe with biochemists that the flocculation of colloids is the essential factor in the preparation of the state of epilepsy. According to Lumière, in epilepsy, as in anaphylaxis, the flocculation of foreign colloids (colloidoclasis), penetrating into the circulation, is at play. The most common path of such penetration is the gastro-intestinal tract. Hustin and Bouché have shown that under the influence of vasculotrophic shock the pyramidal cells in the cortex undergo swelling and chromatolysis. The primary cause of the epileptic seizure might be attributed to excitation by products of flocculation (Lumière). Epilepsy is the result of cortical inhibition secondary to an irritation (Hartenberg).

Chapter XII, Physical Bases of Nervous Reactions: The development of neuromyons, by virtue of which the tropisms become reflexes and reflexes keep the traces of previous reactions, marks the evolution of primary irritability of undifferentiated protoplasm into nervous excitability. The explanation of the real nature of sensations (gustatory, olfactory, visual, etc.) lies in the concepts of physical chemistry (colloidal). This is unquestionable with regard to the gustatory and olfactory sensations. It must be electrolytes that provide the terminal endings of the optic nerve with the means to react to the electromagnetic waves of light. Excitability is the result of the modification of the chemical state and of the osmotic tension of the humoral components. As chemotropism was the driving force behind neurogenesis, so it remained the basic principle in the interneuronal conduction.

Chapter XIII, Electromagnetic Dynamism of Thought: In this chapter, concluding the book, the author develops the premises to the electromagnetic theory of nervous activity. In nerve tissue, more than in any other, the structural complexity became adapted to the progress of functional differentiation. The author contrasts the "neuron theory" of Ramon y Cajal with the "neurite" theory of Apáthy. It is to the latter that the author gives preference. He discusses the view which attributes to the nervous impulse a physical significance. According to this view, the nerve impulse is electromagnetic (bio-electric) in nature. Developing an interesting argument, he comes to the view that between elements of nerve tissue there exist distance-actions; these distance-actions are not chemical but electric. The interneuronal influences are conveyed, not through the processes of secretion, but through the medium of the whole brain; these influences do not act by their specific qualities but by specific energies. "But probability," says the author, "is not certitude, and I do not presume I give the arguments."

Summarizing this work, the aim of which was to show that "in the nervous system there are no centers," the author recapitulates the main points of his thesis. He calls critical minds "to fight a good battle, to react against the tyranny of nerve centers which for so many years has been the game of babblers and has held back many an advancement in neurology."

The book is written with amazing erudition and remarkable skill of argumentation, but when any author covers so many fields it is well to remain skeptical of his accuracy in all cases. For example, the references to colloidal chemistry savor of oracularness. It is all too easy to call in the latest infant science to explain one's own field, so when the author speaks of "distance-actions" as not chemical but electric, the reader feels supported in his skepticism and desires to ask for definitions. Nevertheless, many of the points made are good, and the book is challenging and stimulating; many neurologists will gain a new point of view by reading it.

THE BASIS OF EPILEPSY. By EDWARD A. TRACY. Price, $2. Pp. 92. Boston: Richard G. Badger, 1930.

This book is a restatement, with some additional data, of the observations and theories presented by the author in 1926 and reviewed in the ARCHIVES (**17**:869 [June] 1927). In the foreword a summary of the contents is given: "The first section reports an investigation of chronic epilepsy, major mal cases, made chiefly from 1916 to 1918. This investigation revealed the white spots of epilepsy and the various abnormalities of the vasoconstriction reflexes. These abnormalities appear to be the clinical manifestations of the pathology of the sympathetic in epilepsy previously discovered and described by Echeverria.

"The second section treats of incipient epilepsy, the important phase of the disease which is often present without being recognized until convulsions occur. This investigation began in 1918.

"The third section presents: (*A*) The Vasomotor Reflexes of the Skin, with a method for testing and recording them; (*B*) A Method for Diagnosing Epilepsy Between the Seizures; (*C*) Supplementary notes on the white spots of epilepsy: 1, ordinary; 2, 'new-born'; (*D*) Epilepsy a disease entity."

The first section contains ninety-four observations, made on one patient, of the duration and variability of the "vasoconstriction reaction," i. e., the number of seconds it takes for the white streak to become visible after making the "evoking stroke" on the skin with the "vasomotor tester." It is stated that "a most interesting relation was discovered between the reaction-times and the onset of convulsions. A daily record of the reaction-times and the hours when convulsions occurred during a period of two years revealed that a lessening of one or more of the reaction-times took place within twelve hours before the onset of convulsions. Since a lessening of the reaction-time indicates an increase of irritability in the sympathetic neurons involved in the reaction, the records indicate an increase of irritability in sympathetic neurons preceding a convulsion." The first fifty observations are presented in the text (pages 15-19) to show that the variations occur "apparently without rule or reason," and the next forty-four are given on pages 20-25 with notes showing their relationship to the onset of a convulsion, usually about eight hours before the seizure, but a few were within minutes of the onset and some were as long as thirteen hours before. The data are so presented that it is difficult to analyze them, but a close study shows that some observations (e. g., "Obs. 585") are used in both places, and others used in the first category (e. g., "Obs. 271") are even nearer the onset of a convulsion than other observations used to show the relationship to convulsions. Even ignoring such misuse of data, the figures are not striking. Only if unselected and tabulated, so that they could be statistically treated, would they be significant.

The other point emphasized is "That in the normal individual, the reflex vasoconstriction (the white streak) to stroking the skin of the forearm appears sooner than that which follows stroking the cheek. In chronic major mal the reverse is

frequently noted, i. e., the reaction-time of the cheek is less than that of the forearm." All of these observations are made practically worthless by the absence of control data from normal persons.

The autopsies by Echeverria, published sixty years ago, are reviewed by the author and put forward as important evidence favoring the sympathetic etiology of epilepsy. It would have been useful to have quoted also the more recent pathologic work of Herzog (Histopathologische Veränderungen im Sympathicus und ihre Bedeutung, Deutsche Ztschr. f. Nervenh. **107**:75, 1928) and Laignel-Lavastine (Pathologie du sympathique, Paris, Félix Alcan, 1924) which show that such microscopic changes as those described by Echeverria are common and probably not important.

The illustrations, which reproduce photographs of the "white spots" and of the vasomotor reaction of the skin, are not convincing for differences in the darkness of the printing would seem to account for some of the differences described as physiologic (compare plates A with AA), and the "white spots" shown in plates XII and XIII look like technical artefacts. Altogether the book gives a poor impression. Although the theory that epilepsy is due to "diseased fibres of the vegetative nervous system" is interesting, the evidence is presented in so unsystematic a manner and the discussion is so lacking in critical judgment, that the reviewer is left unconvinced. Nothing convincing is presented to explain the basis of epilepsy.

THE ADAPTATION OF THE EYE: ITS RELATION TO THE CRITICAL FREQUENCY OF FLICKER. By R. J. LYTHGOE and K. TANSLEY. Medical Research Council, Great Britain, Special Report No. 134 from the Committee upon the Physiology of Vision. Price, 2 shillings, 6 pence. Pp. 70. London: His Majesty's Stationery Office, 1929.

This work is divided into two parts and an appendix. Part 1 discusses the critical frequency of flicker during the course of light and dark adaptation. Part 2 considers the influence of the surroundings on the critical frequency of flicker. The appendix discusses the light threshold during dark adaptation with special reference to a suspected case of night blindness. The bibliography is complete.

Quoting in part from the preface: "a knowledge of the behavior of the eye in different states of its adaptation is obviously of great importance for all occupations involving vision at low illuminations, and within recent years it has become recognized that adaptation may also play a prominent part in the performance of visual tasks at higher illuminations, since it is then controlled chiefly by the characters of the surrounding field of vision. Those physicists who have used the flicker method have recognized that physiological factors have an important influence on the results, but the matter has not been carried farther. The present report shows the importance of a physiological outlook on the part of those using this kind of measurement. Its publication appears also to the Committee to be justified not only by the decisive nature of the results obtained, but also by their theoretical interest."

The sensation of vision persists for a certain time after visual stimulation has ceased. Very early in the history of physics, attempts were made to measure the frequency of visual stimuli necessary for the persistence of a sensation of continuous vision. The use of the critical frequency of flicker is a refinement of any previous attempts. The experimental work in this essay considered this problem in an eye during the course of dark adaptation, in the almost completely dark adapted eye, and during the course of light adaptation. Foveal stimulation and response was of necessity compared with peripheral retinal responses from similar stimuli. The brightness values of surroundings and of test-patch is proved to be of basic importance.

Many different observations arose during the course of these experiments, some of them rather new, others corroborating the work of previous investigators. This seemed especially so in part 2 of the work. Naturally, in a review of this nature it is impossible to include all such desiderata in the summary of the results

obtained through the experimental work. Also a description of the apparatus used, the methods of procedure, the interpretation of the observations and the part which the results of previous investigations played in the conclusions of these authors cannot be explained in abstract.

As a result of this experimental work: "(a) the critical frequency of flicker measured for foveal and peripheral retina under those adaptation conditions previously mentioned herein, and (b) the influence of the brightness of the surroundings upon this critical frequency of flicker for illuminations of the test-patch by different intensities of white and red light," the following conclusions were reached by the authors. The critical frequency due to cones falls during dark adaptation and with decreasing levels of dark adaptation, and it is highest when the brightness of the test-patch and the surroundings are equal. The critical frequency due to the rods rises both during dark adaptation and with decreasing levels of light adaptation, and is highest with completely dark surroundings. The peripheral cones are functionally not identical with the foveal cones. The brightness of the surroundings is the most important factor in determining whether critical frequency relations are of the rod or the cone type, bright surroundings encouraging the cones and dark surroundings the rods. At intermediate illuminations of the test-patch and surroundings, the resultant critical frequency appears to be due partly to rods and partly to cones. The results obtained by the critical frequency of flicker method are in good agreement with the performance of the eye under other visual tests. The Ferry-Porter law (a ratio expression which considers the number of flicker flashes a second at the critical frequency with the illumination of the test-patch) holds at 10 degrees on the peripheral retina when the brightness of the surroundings is the same as is that of the test-patch.

All the tables of the results of these experimental methods and from which the authors saw fit to state the conclusions found follow after the appendix.

LES PLEXUS CHOROIDES. By DR. NATHALIE ZAND. Préface du Professor Roussy. Price, 22 francs. Pp. 137. Paris: Masson & Cie.

In this monograph Mme. Zand discusses in some detail the anatomy, physiology and pathology of the choroid plexuses. None of these topics seems to her to be settled, especially the one pertaining to the physiology. While Zand accepts the choroid plexuses as the place of origin of the spinal fluid, she still considers unsettled the question whether the fluid is a dialysate or a product of secretion by the epithelial cells. Small amounts, however, are also contributed by the ependyma and the pial blood vessels. Another function of the choroid plexus stressed by Zand is protective — it removes harmful substances from the blood serum. This is accomplished by the leptomeninges and the reticulo-endothelial cells (histiocytes) which, though scarce in a normal choroid plexus, sometimes appear in the cerebrospinal fluid. In the choroid plexus and pia they are situated between the vessel walls and the epithelium; in the brain substance they are closely adherent to the adventitia. Concerning the function of the cerebrospinal fluid, Zand thinks that it provides the nervous system with "ideal nutritive as well as mechanical conditions," a view somewhat analogous to that of Monakow—that the cerebrospinal fluid supplies the nervous system with necessary nutritive substances and rids it of useless products of disintegration. Many other equally unfounded views are quoted, derived generally from morphologic and experimental but not pathologic studies.

In speaking ot the pathology of the choroid plexuses, Zand states that they show changes in tumors of the brain, dementia praecox and infectious or degenerative conditions. The changes are acute (mainly degeneration of the epithelium) and chronic (hyperplasia of the stroma) in alcoholism, dementia praecox and arteriosclerosis. Entirely normal choroid plexuses are, according to Zand, very rare, for they easily react to any morbid condition that may befall the human body. For this reason the choroid plexuses are especially affected in old age when they show the imprints of the various morbid conditions that had occurred during life plus the changes caused by old age itself. Yet to such an organ, usually diseased, is ascribed the function of production of the spinal fluid.

The few instances quoted show how prevalent are the hypotheses in Zand's monograph. The latter reflects the attitude of many workers toward the various problems pertaining to the choroid plexus. The book is of some value as it undoubtedly will arouse interest in this structure that has been so much neglected both by the general pathologist and by the neuropathologist.

THE LANGUAGE DEVELOPMENT OF THE PRESCHOOL CHILD. By DOROTHEA A. MCCARTHY, PH.D. University of Minnesota Institute of Child Welfare, Monograph Series no. IV. Price, $2.50. Pp. 174. Minneapolis: University of Minnesota Press.

This important monograph on the language development of the preschool child is one of a series of valuable books appearing under the auspices of the Institute of Child Welfare of the University of Minnesota. The reviewer is in hearty agreement with the opinion expressed in the foreword written by John E. Anderson, Director of the Institute, that this monograph stands out as one of the best studies of the development of language. He says further that "previous studies of the development of language in early childhood, have, as a rule, been concerned with one or two children or with selected groups. In the study that is here presented, Dr. McCarthy has taken unusual precautions to secure a group representative of the general population by selecting her children within each age level on the basis of socio-economic status. Further control has been secured by obtaining an equal number of responses for each child, by limiting the age groups to children within a month of the age specified, and by equating the number of individuals in each age and sex group.

"The analysis of the data is unusually complete. In addition to a study of the length and structure of the sentence and of the use of parts of speech, an interesting modification of the Piaget functional analysis has been used. The relationship of various language processes to age, sex, socio-economic status, intelligence, position in family, number of playmates, and other factors has been investigated."

A number of direct results were obtained. One was a clearcut difference indicating a more rapid development of language among girls and an early language development among the children of the upper social economic class. The most striking result, however, was in the fact that a child at 18 months knows only a few single words, and yet in a short time — three years — he has acquired several thousand words which he is able to combine into sentences and use with the same complexity in his everyday conversation that an adult is able to do. This indeed is a surprising observation.

The book is of the utmost value to those who are interested in the development and study of speech.

THE ACTION OF MUSCLES INCLUDING MUSCLE REST AND MUSCLE RE-EDUCATION. By SIR COLIN MACKENZIE, M.D., F.R.C.S., F.R.S. (EDIN.). Second edition. Price, $3.50. Pp. 288. New York: Paul B. Hoeber, Inc., 1930.

A knowledge of the action of muscles and their reeducation following disease is of primary interest not only to the orthopedic surgeon but also to the neurologist. This excellent book, which originally appeared in 1918, has been revised and a second edition has just been issued. It differs little from the first with the exception that there is a special section dealing with erect posture with which every system in the body is correlated.

The book begins by a statement of principles. This part, which includes fifty-five pages, is an excellent presentation of how muscle paralyses should be tested and how treatment should be applied. For example, the author makes the statement that in poliomyelitis no massage or electricity should be applied until recovery has taken place. This no doubt will come as a shock to most neurologists and orthopedic surgeons who are accustomed to order one or both methods of treatment as soon as such a patient comes under their care. The author states

that paralyses of muscles rarely occur, and that in most diseases which produce paralysis there is only a partial loss of function, and he shows how muscles can be tested so that their accurate status can be determined. He makes one very interesting statement. He believes that the thymus gland has a direct relation with the growth of muscle and recommends its use in the early stages of poliomyelitis during reeducation, and similarly during the reeducation of weakened muscle from nerve or other injury. The question is suggested whether in some remote way this gland is associated with the failure in man of a limb to regenerate after amputation — a power which our remote ancestors before the evolution of the mammalian type appear to have possessed in conjunction with a highly developed reflex system.

Altogether this is an excellent book and should be in the hands of every neurologist and orthopedic surgeon.

HUMAN BIOLOGY AND RACIAL WELFARE. Edited by EDMUND V. COWDRY, with an introduction by EDWIN R. EMBREE, with contributions by twenty-eight authors. Price, $7.50. Pp. 612. New York: Paul B. Hoeber, Inc., 1930.

The very contemplation of this title and the names of the distinguished authors is sufficient to inspire the belief in the reader that here is an outstanding book, one that will fill in the gaps in the all too specialized knowledge, and give one an idea of man as a whole. As it is, the book has the strength and the weakness of such a project. Its greatest failing is the unevenness of the contributions. Some of them are written in language so simple that it would bore the average educated reader. Other chapters are written with a wealth of technical detail that could not be mastered without a rather extensive preparation in the fields they are designed to cover. As a series of essays on the general field, most of them are admirable. As a résumé of man's knowledge of himself, the book leaves something to be desired. Although many of the chapters are written by distinguished medical authors, it must be admitted that nonmedical scientists have put forward a more succinct and generally readable series of essays than have physicians. Certain chapters are outstanding, notably the first, entitled "Life in Space and Time," by Henry Norris Russell; but also "Mental Evolution in the Primates," by Robert M. Yerkes; "Human Races," by Ales Hrdlicka; "The Integrative Action of the Vascular System," by Walter B. Cannon; "The Effect of Climate and Weather," by Ellsworth Huntington; and "The Relation of Science to Industry," by R. A. Millikan.

Dr. Cowdry has attempted in this single volume to synthesize the different aspects of man in a manner analogous to his treatment of the cell in his "Cytology." The purpose is excellent. There is need of such a synthesis, but the product is incomplete. The greatest deficiency is in the realm of psychiatry and analytical psychology, for human motives and mental mechanisms are nowhere adequately stressed. It may be that these deficiencies merely serve to call attention to the general excellence of the volume, and it must be admitted that the reviewer was disappointed to turn over the page and find himself in the index with no more worlds to conquer. Handsomely printed and well illustrated, the book makes an immediate appeal, and in sketching on broad lines the front of advance of human knowledge it forms a most unusual background on which one can project for his own delectation the position and aims of the specialized branch of knowledge to which neuropsychiatrists are devoted.

NEUROLOGY. By KNUD H. KRABBE. Spanish translation by S. Pi Suñer, Pp. 368. Barcelona: M. Marín, 1929.

Rather than a treatise on nervous diseases, Krabbe's book, originally published in Danish, is a useful guide to the study and diagnosis of the most important nervous disorders. Since each of the diseases considered is described at length their diagnosis by the student and the general practitioner — for whom the book

was primarily written — is greatly facilitated. This is true in spite of the fact that the book is not illustrated, only three diagrams being included in the text. However, the lucid style in which it is written compensates for the lack of illustrations.

The first chapter deals with the neurologic examination of the patient. The most important symptoms and their diagnostic value are discussed, and the value of the diverse tests is emphasized. In successive chapters, the acute and chronic inflammatory diseases of the central nervous system and their sequelae are examined. Treatment in these diseases is also discussed. This is followed by a study of the myelopathy of pernicious anemia and such diseases as polyneuritis, mononeuritis, neuritis of the cranial nerves, neuritis of the limbs, causalgia and rheumatic muscular diseases.

The tumors of the central nervous system and their varied symptoms are the subject of a chapter, followed by another section on the vascular disturbances of the brain and spinal cord. The traumatic lesions of the central nervous system, and the hereditary and nonhereditary abiotrophic nervous diseases are in turn described and discussed. The last three chapters deal with the paroxystic nervous diseases, neurasthenia and hysteria, respectively. A list of the most important treatises and monographs from the point of view of their value to the student and general practitioner ends the book.

OLD AGE THE MAJOR INVOLUTION. THE PHYSIOLOGY AND PATHOLOGY OF THE AGING PROCESS. By ALDRED SCOTT WARTHIN, PH.D., M.D., LL.D. Price, $3. Pp. 185. New York: Paul B. Hoeber, Inc., 1929.

There are few books written on old age. In common with most human beings, medical men seem to avoid the subject. This book therefore is timely. The author considers old age as a part of the normal physiologic process. To him life is divided into evolution, maturity and involution. Human beings in common with other living species are born to propagate themselves. Once they are through with this physiologic process there is no more excuse for them to live and therefore death is inevitable. Instead of dying promptly, however, as one should, human beings take their time about it, and this is old age. This is not at all depressing, for after all why should one wish to live after reaching old age?

From the medical standpoint it is an excellent presentation of the physiologic functions which are present in the evolution of the human species, but chiefly the book is notable in that it summarizes the pathologic changes which occur in old age. It is not a book to be read by youth, for youth does not understand anything but itself. One has to be old to understand this cheerful philosophy.

PROBLEM TENDENCIES IN CHILDREN. A METHOD FOR THEIR MEASUREMENT AND DESCRIPTION. By WILLARD C. OLSON, Price, $2. Pp. 92. Minneapolis: University of Minnesota Press, 1930.

This book reports an attempt to determine problem tendencies in children by the use of a rating scale. The first part of the book describes the method. Two schedules are used: one consists of a record of specific overt acts of misbehavior, and the other of a rating in personal traits. The two are related statistically so that the rating in the child's characteristics gives a score that serves as a problem-tendency indication. The author applied this rating scale to the pupils of fifteen schools and concluded that the validity of the score has been supported by clinical studies and by the subsequent testing of the children. The remainder of the book is devoted to an analysis of the results in terms of such variables as age, grade, sex, achievement and intelligence. The ratings were affected by or related to sex (boys having a higher degree of problem tendency than girls), intelligence (in each grade the chronologically average child and mentally underage child were the greatest problems) and school achievement (lack of school achievement giving a high degree of problem tendencies).

An ample appendix is devoted to details concerning the schedules and the methods of scoring.

DIE ARTEN DER SCHLAGANFÄLLE DES GEHIRNS UND IHRE ENTSTEHUNG. By P. SCHWARTZ. Price, 48 marks. Pp. 269. Berlin: Julius Springer, 1930.

The first part of this monograph is given over to a thorough consideration of the various ideas concerning the pathogenesis of apoplectic attacks. Embolic brain insults, arteriosclerotic thromboses and hemorrhagic insults are all reviewed. In the second portion the author presents the results of pathologic study in many cases of cerebral insults due to embolic and hemorrhagic causes. The work is finely illustrated, and the photographs are well chosen. The analysis of the case material is well done. The monograph is an excellent review of the subject of apoplectic attacks and should be read by all neurologists who wish to keep abreast of the latest ideas in this field.

CHILDREN AT THE CROSS ROADS. By AGNES E. BENEDICT. Price, $1.50. Pp. 238. New York: The Commonwealth Fund, 1930.

The object of this book is to depict in popular manner the work of the visiting teacher in a rural community; the author has accomplished her object well. The book is divided into four parts. In the first two, community, home and school factors causing problem behavior in children and treatment measures found adequate in individual cases are presented in story form. The last two parts deal with the special problems a rural community presents to the visiting teacher. The whole is presented in a clear, nontechnical form. It should be read by every teacher, visiting nurse and attendance and probation officer, both rural and urban, and should form part of the library of every school.

TECHNIK DER MIKROSKOPISCHEN UNTERSUCHUNG DES NERVENSYSTEMS. By DR. W. SPIELMEYER, Professor an der Universität München. Fourth edition. Price, 11.80 marks. Pp. 168. Berlin: Julius Springer, 1930.

The fourth edition of Spielmeyer's well known Technik for the most part contains the staining methods that have been standard for years. It also contains newer methods relating particularly to the glia. It is manifestly impossible to collect all the methods of staining that have appeared, particularly within the last five years. Spielmeyer has made no effort to include every recently described method, but has wisely selected those that have proved of value in his own laboratory. The Hortega method is well given, but only a few of the innumerable modifications are described. He gives, particularly, the modifications of Metz and Spatz, Globus, Penfield and Kanzler. Neuropathologists are greatly indebted to Spielmeyer for keeping this book up to date. It should be in every laboratory in which nerve tissue is examined.

A POINT SCALE OF PERFORMANCE TESTS. VOLUME I. CLINICAL MANUAL. By GRACE ARTHUR. Price, $1.50. Pp. 82. New York: The Commonwealth Fund, 1930.

This is a manual of directions and scoring to use in giving a standardized scale of performance tests. Such a nonverbal scale is needed for cases in which the highly verbalized Binet tests are inadequate because of a foreign language handicap, speech or hearing defect or the presence of a marked irregularity in the development of verbal and nonverbal abilities. It is to be hoped that this scale will fill the need.

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